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THE MANAGEMENT OF FRONTAL SINUSITIS AND ITS COMPLICATIONS

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THIS is a large subject and I have tried to select certain aspects of interest. An enormous literature on this subject has accumulated during the past 60 to 70 years, but I have found greater value in reading 30 years and more of the case histories of patients admitted to the Royal Victoria Infirmary. Throughout the latter part of this paper the case histories speak for themselves and I can do little more than comment on them.

It is well known that the majority of patients suffering from acute frontal sinusitis recover with little more than supportive measures, helped at times by antibiotics. Of course, the right antibiotic or chemotherapeutic agent must be given in adequate dosage. Those patients recovering with minute doses of antibiotics did not need them. The bulk of these patients never attend hospital and in general it is only the severe cases of frontal sinusitis and its complications which are seen in hospital practice. Even so, most cases of uncomplicated acute disease recover with adequate dosage of the right chemotherapeutic agent, usually penicillin possibly combined with a sulphonamide or streptomycin. Prior to antibiotic therapy, drainage of the frontal sinus by removal of the anterior end of the middle turbinate and possibly bouginage formed the usual and successful practice in curing these lesions. Today such measures may have occasional application. Better still is the alternative of trephining the floor of the frontal sinus so as to keep away from

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the fronto-nasal duct and avoid delay in the recovery of its function. Vacuum headache or pain due to a lesion in the sinus or its duct (as distinct from migraine which is readily confused with this) arises from a mild catarrhal inflammation producing partial obstruction in, and ciliary failure of, the fronto-nasal duct from œdema of its lining mucosa

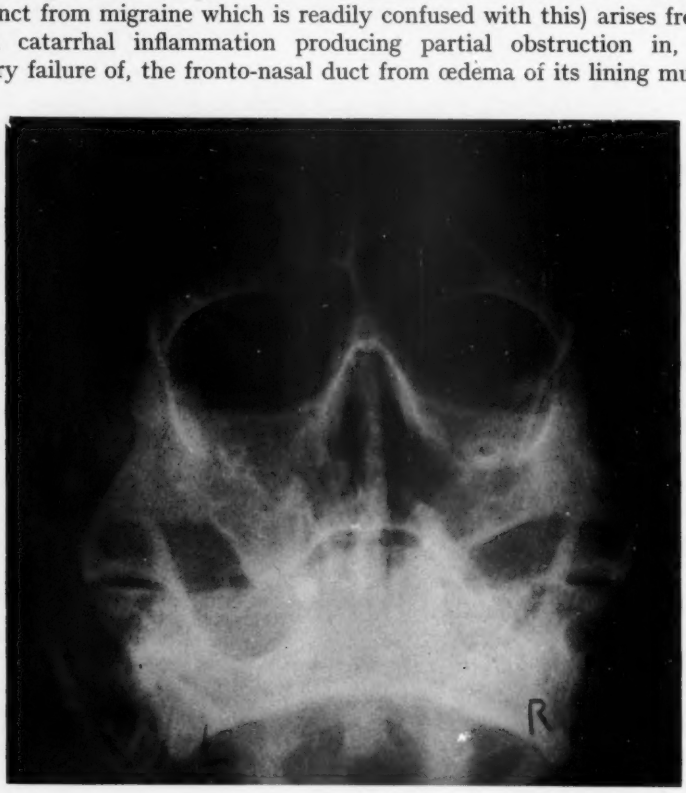

FIG. 1.

Radiograph showing opaque frontal and maxillary sinuses. The nasal antrostomy openings made by a Caldwell-Luc approach some years earlier can be seen.

and intermittent obstruction by mucoid or mucopurulent secretions from the sinus. An attack clears with the same simple measures as are used in acute frontal sinusitis. I regard this as an acute form of frontal sinus disease and as a lesion which may be recurrent.

Repeated attacks of acute frontal sinus disease may be due to true re-infection, at times associated with persistent obstruction of the duct caused by chronic disease in the neighbouring sinuses.

Fig. 1 illustrates a case of recurrent frontal sinusitis associated with chronic maxillary infection in which there was an interval of 7 years between exacerbations. Achromycin therapy with repeated antral lavage

sufficed on this occasion, but she had had a Caldwell Luc done at a much earlier date for a similar attack.

Another important factor in recurring frontal sinusitis is mechanical obstruction of the duct, for example an osteoma in the ethmoids or frontal sinus (Figs. 2 and 3) when removal is indicated. Ethmoidal polypi, allergic or suppurative in origin, or a mucoid or purulent cyst of

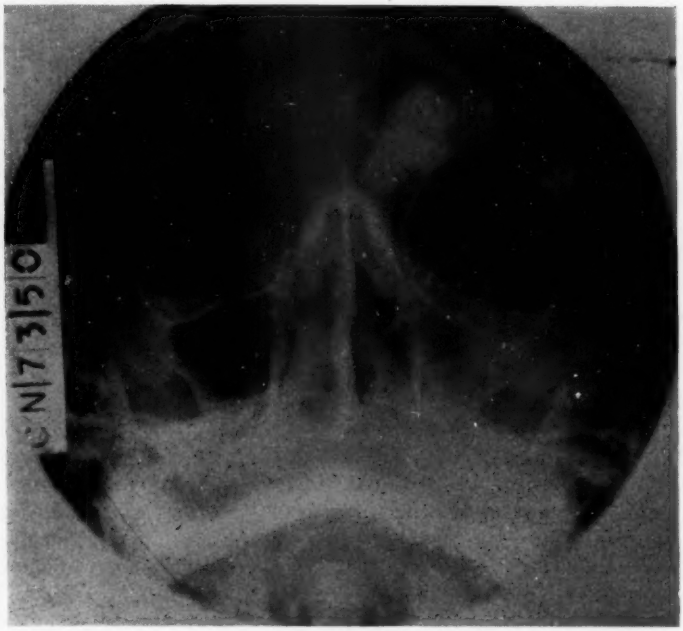

FIG. 2.

Radiograph to show an osteoma of the frontal sinus partly obstructing one fronto-nasal duct. Opacity of both frontal sinuses and one maxillary sinus was due to an acute multi-sinusitis.

the middle turbinate may cause persistent œdema and mechanical obstruction of the fronto-nasal duct. Each of these should be dealt with by intranasal, transantral, or external operation according to the extent of the disease and the preference and judgement of the surgeon. A septal deformity is also shown with compression of the middle turbinate on one side and a so-called hypertrophy of the other. Mere correction of the septal deformity rarely prevents further trouble in the frontal sinus and the deformity itself would not appear to be a frequent cause of mechanical obstruction in the duct. In such cases polypi or granulation

tissue are frequently found within the middle meatus at the fronto-nasal duct exit, and the supposed hypertrophy of the turbinate is revealed as a mucocœle or pyocœle. Submucous septal resection may be necessary for access to the ethmoidal region but let us not imagine that a septal deformity narrows the fronto-nasal duct or interferes with its ciliary function. Of course, the acute frontal sinus lesion may in fact be an exacerbation of a chronic condition.

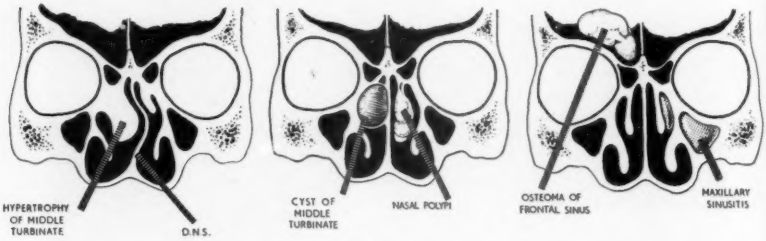

FIG. 3.
Diagram to illustrate mechanical and infective factors producing obstruction of the fronto-nasal duct.

Intranasal procedures thus far described have been related to infection in sinuses other than the frontal, and the only surgery directly interfering with turbinal tissue has been a limited removal of the anterior end of the middle turbinate for access to disease at the exit of the fronto-nasal duct. Every effort has been made to preserve the function of the duct. Many intranasal procedures have been designed for reconstruction or destruction of the fronto-nasal duct so as to provide wide drainage from the sinus through its floor: such procedures lead to a varying degree of destruction of the middle turbinate and also the ethmoid labyrinth. Operations of this kind were indicated when the ethmoids were full of polypi and pus and when cure of other associated lesions failed to produce relief of pain and persistent purulent discharge from the frontal sinus. The fronto-nasal duct and the ethmoids were held to be at fault and extensive intranasal techniques were designed to excise all the disease. Rasping, drilling, mucosal flaps and extensive ethmoidectomies such as those of Mosher (1913) and Sluder (1919) were popularized (Fig. 4). Many surgeons found these difficult, and the catastrophe of damage to the roof of the ethmoidal labyrinth and cribriform plate was a surprisingly common occurrence in hands less skilled than those of the originators. Only one post-operative case of meningitis following removal of polypi occurred in the department in the past 30 years and that was in 1933. Damage to the lamina papyracea and the optic nerve have been reported but only one example of injury to orbital fat has been found

in our records and this occurred during an enthusiastic intranasal removal of polypi. Perhaps more important, the new ducts often filled with granulation tissue, scar tissue and new bone formation, pus persisted in the ethmoids and more extensive surgery might be attempted by the external approach, often failing and leaving the surgeons either in despair

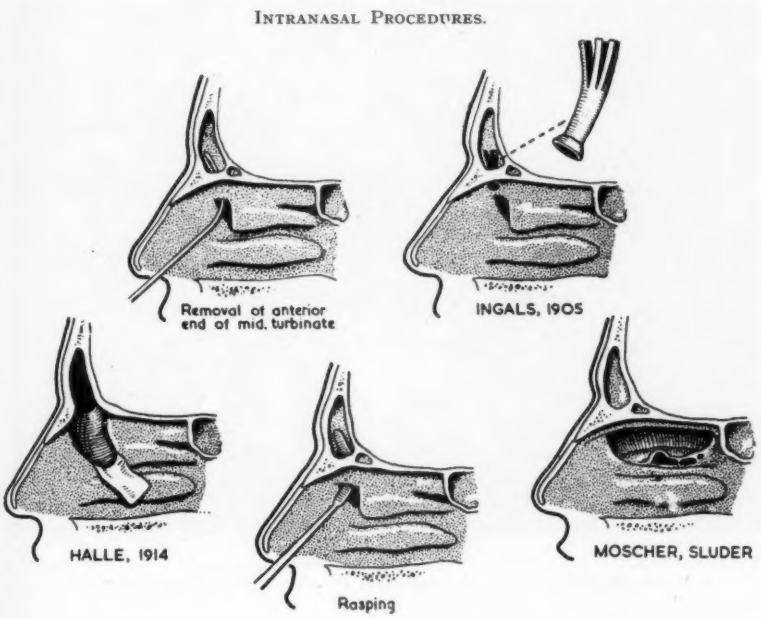

Fig. 4.
Diagram illustrating some intranasal operations on the frontal and ethmoid sinuses.

or willing to accept a persistent discharge without pain as a reasonable result. The lack of respect for turbinal tissue produced wide nasal airways full of crusts and lined by atrophic mucosa. The patient was often worse off than before.

Stenosis of the new duct was frequently due to persistence of uncontrolled infection, and spreading osteitis was a dreaded complication. When limited intranasal procedures failed some surgeons undertook extensive ethmoidectomies by an external approach (see Fig. 5).

The wide variety of approach to the problem was surely an index of dissatisfaction. Persistent uncontrolled infection still caused trouble. Gradually it was learnt that where a fronto-nasal duct no longer functioned well the best procedure was a drainage operation of the type described

by Howarth (1921) and Lynch (1921,) designed to leave the frontal sinus mucosa intact. Where the ethmoids were dealt with by an external approach a better view of the frontal sinus and ethmoidal labyrinth was obtained and the middle turbinate could be largely preserved despite extensive excision of disease. Stenosis of the new duct might still recur.

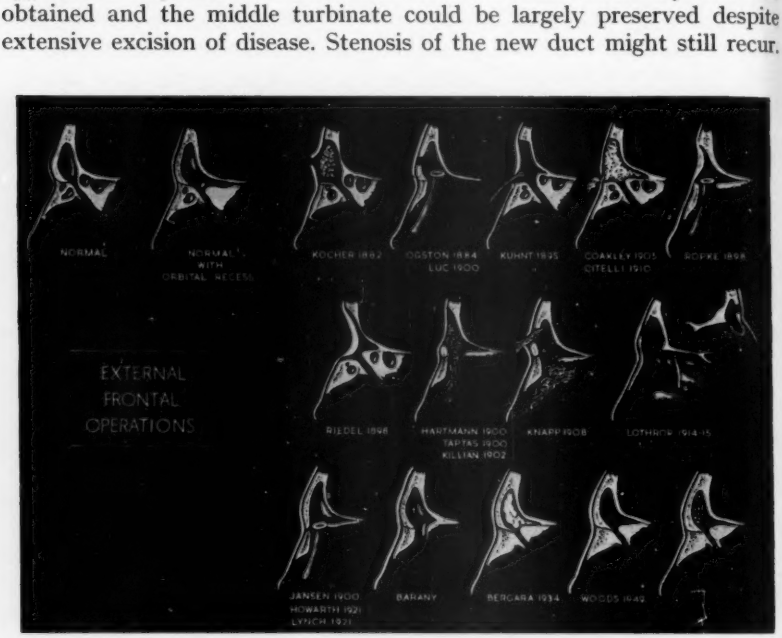

FIG. 5.
Diagrams to illustrate some examples of external frontal operations.

Skin grafting of the new duct, and prolonged intubation with inert plastic tubes and, more important, the control of infection by antibiotic therapy helped to solve the problem, in particular when the infected frontal sinus mucosa was capable of a full recovery. Bárány's (1924) operation, where a mucosal lining of the new drainage opening is preserved, appeals to me in principle but I have no experience of it. However, although infection is more likely to be controlled in the present era, patients with chronic frontal sinusitis may have a grossly diseased mucosa, irreversibly damaged, something no better than a "pus factory". Under these conditions, total removal of the mucosa is indicated in the hope of obliteration of the cavity by scar tissue and bone. Indeed, by using the technique of Woods (1951 and 1955) and Bergara (1955), it is hoped that the fronto-nasal duct may also be obliterated. Other deforming obliterative procedures where the anterior and inferior walls of the frontal sinus were removed together with drainage and packing

of various sorts failed in some instances because the configuration of the sinuses is such that soft tissue obliteration is almost unattainable. External drainage operations failed because of loculation of pus and stenosis of the duct from uncontrolled infection or irreversible disease of the mucosa. Obliterative procedures fail if any mucosa is left behind since this may later produce a mucocœle or pyocœle. It is true that total removal requires considerable skill but it is possible to attain it. Woods

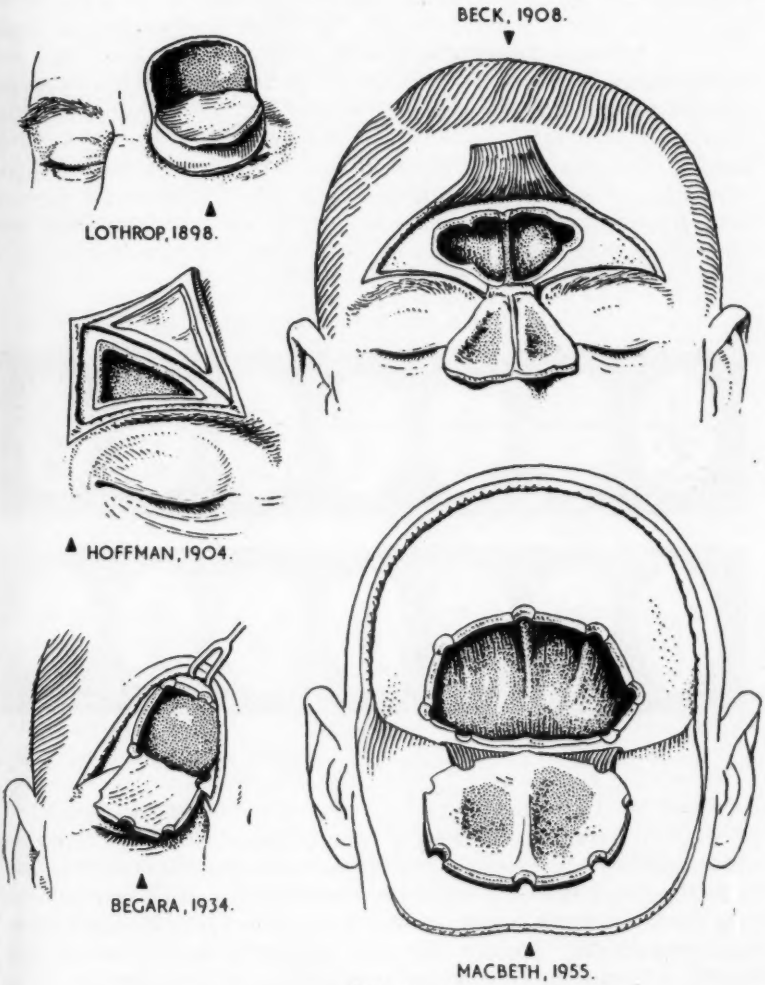

FIG. 6.
Diagram illustrating the osteo-plastic approach to the frontal sinuses.

excises mucosa through the floor by using gauze controlled by small mirrors. Bergara (1955), Macbeth (1954) and others employed an osteoplastic frontal flap (Fig. 6). The former method is useful in small sinuses and the latter in all sinuses. The osteoplastic flap has a long history. It gives wide exposure and makes mucosal stripping easy; but presumably the technique fell into disuse because of the danger of producing osteitis of the frontal bone by cutting through the cancellous bone in the anterior wall, and the opening of diploic tissue from within the sinus when a curette was used to remove mucosa. Curettage readily opens up diploic tissue by breaking down the very thin cortical plate (100μ-300μ) separating mucosa from diploic tissue. Although I favour Bergara's approach through the eyebrow as being useful only for the majority of frontal sinuses, the large osteoplastic flap as used by Macbeth (1954) has advantages in the exceptionally large sinus. Certainly we cannot complain of the lack of operations, or indeed of the lack of incisions for approach to the frontal sinus (Fig. 7). Yet an odd patient exists in every department who continues to have trouble and the question of eradication arises.

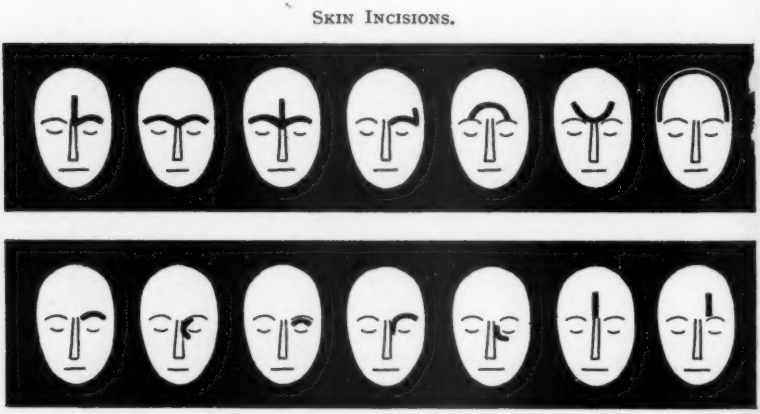

Fig. 7.
Diagram to show the incisions of approach to the frontal sinus.

Table I shows an attempt to summarize my views on acute and recurrent frontal sinusitis. I believe that intranasal procedures should be directed against associated lesions and there should be no interference with the fronto-nasal duct other than at its exit. The middle and inferior turbinates should if possible always be preserved. In the case of chronic frontal sinusitis, the assessment of reversible and irreversible damage in a large proportion of cases will depend on the surgeon's experience

(Table 2). We know that damage is irreversible when a good drainage operation fails; this is not something the pathologist can tell us.

TABLE I.

ACUTE FRONTAL SINUSITIS.

1. Conservative measures and antibiotics when necessary.
2. Surgery limited to trephining the cortical bone of the floor of the sinus.

RECURRENT FRONTAL SINUSITIS

1. Obstruction of the fronto-nasal duct.
   (a) Mechanical—Polypi, cyst of middle turbinate, D.N S.
   (b) Infective—Ethmoiditis or Maxillary Sinusitis.
   (Maybe a combination of Mechanical and Infection factors)
   (c) Re-infection—treat each new attack as it arises.
   (d) Acute exacerbation of a chronic frontal sinusitis.

The fronto-nasal duct and turbinates must be treated with great respect.

TABLE II.

CHRONIC FRONTAL SINUSITIS.

When mucosal changes are reversible a mere drainage operation will suffice and the frontal sinus mucosa is respected.
If the mucosal changes are irreversible then the sinus mucosa should be removed in its entirety.
Always respect turbinal tissue.

Many complications of frontal sinusitis are of acute onset and may arise from acute sinusitis or from an acute exacerbation of chronic disease. Even the chronic erosive lesions of the anterior wall or mucocœles frequently present acutely. As a theme I have therefore selected those cases presenting as acute "ruptured frontal sinuses". Where the complications of frontal sinusitis are of significance in the absence of acute signs these are discussed under separate titles. The term "ruptured frontal sinus" has been used when an acute external swelling has been obviously present (Fig. 8). Two main types occur (a) those which rupture through the floor into the orbit (so frequently labelled in textbook photographs as examples of acute sinusitis) and (b) those which rupture through the anterior wall. Undoubtedly some lesions rupture through the posterior wall but not so obviously. The floor readily ruptures because the bone is so thin. Although there is no bony dehiscence at first, a swelling or subperiosteal abscess in the orbit arising as a direct result of frontal sinusitis indicates an osteitis. Granted this is not clinically osteomyelitis but today we prevent spread whereas in the past the patient's natural defence mechanism was alone available. Today, cases progressing from this stage to an osteomyelitis have done so because of improper management by the doctor or failure by the patient to follow instructions. Many

of these cases are primarily referred to the ophthalmologists and frequently have not been regarded with sufficient seriousness by the general practitioner or by the patient in the early days of the infection. Heavy dosage with penicillin, and possibly streptomycin is necessary (for the majority of lesions are due to a pyococcus) and many recover without surgery. The

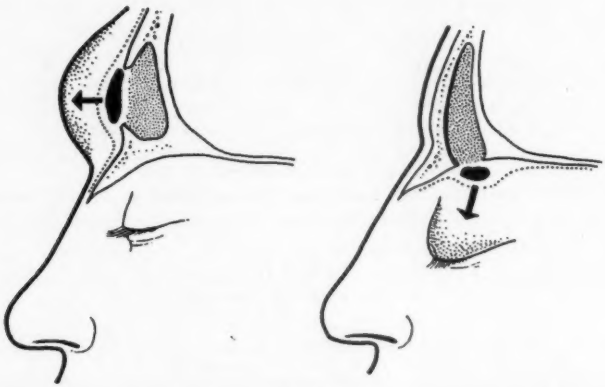

FIG. 8.
Diagram to show the two main types of "Ruptured Frontal Sinus".

two patients (shown in Figs. 9 and 10) were cured by penicillin. Any suggestion of failure to respond rapidly to 1 mega of penicillin 6 hourly together with ½ gram of streptomycin twice daily requires incision and trephining of the floor of the frontal sinus to release pus under pressure. A bacteriological swab should be taken at the time of the operation and antibiotics changed if there is need. Under no circumstances must the external drain be removed until the surgeon is certain that the fronto-nasal duct is functioning adequately as tested by a dye; even then antibiotics must be continued for 4-6 weeks to prevent relapse. Once the acute phase has settled, all the paranasal sinuses must be carefully assessed and a decision made as to whether there is need to tackle associated maxillary or ethmoid lesions by a transantral or intranasal approach. The need for an external operation depends on the degree of functional recovery in the fronto-nasal duct. If the duct fails to work the patient probably has an infected mucocœle or pyocœle.

The patient shown in Fig. 11 was referred from the Eye Department in September 1955 with a one year history of diplopia and proptosis. Pus was washed out of the antrum and an ethmoidal polyp removed. However, he required a combined transantral and intranasal ethmoidectomy from which he appeared to make a satisfactory recovery. Eight months

FIG. 9.
A ruptured frontal sinus which recovered on penicillin therapy.

FIG. 10.
Case of Mr. R. Thomas—recovered fully with penicillin therapy.

later he returned with an infected mucocœle (Fig. 12) and an external frontal operation was performed. Since then he has remained well.

(11) (12)

FIG. 11.

Patient as he presented in September 1955—notice the displacement of the eyeball.

FIG. 12.

Same patient as Fig. 11 when he returned to the Department eight months later with a pyocœle.

Of the 14 cases of mucocœle seen in the department during the past 30 years a large proportion attended hospital because of an acute incident with orbital rupture (Table III). The majority of these had eroded the floor or the anterior wall of the frontal sinus. In one the posterior wall was eroded and dura exposed, and in another the mucocœle communicated with a meningocœle. The early records gave little clue to the extent of disease in the maxillary and ethmoid sinuses but the history frequently suggested a long-standing sinus infection and in the later records there was frequent evidence of such infection. Only 2 cases had a history of injury and I may add that I have never felt egg-shell crackling. Intranasal examination occasionally reveals a mucocœle or pyocœle presenting lateral to the middle turbinate.

Pyocœles are said to be infected mucocœles—but they are indolent purulent collections in the ethmoid or frontal sinuses with the nasal

TABLE III.

Mucocœle.	14 cases
	2 followed injuries (20 years and 43 years previously)
	1 communicated with a meningocœle
	1 recurred 2 years later as pyocœle
	1 exposed dura.
Operation	11 External. 3 Intranasal
Pyocœle	23 (16 appeared as R.F.S.)—bilateral in 2 cases
	Fistula 6—3 followed incision and drainage
	Dura exposed 3
	Sequestrum 1
	C.S.F. leak 1
Operation	20 External. 3 Intranasal

outlet obstructed by swollen mucosa, polyps or bone produced by chronic multisinusitis. Repeated acute exacerbations occur and settle with release

FIG. 13.
Patient presenting with a mucocœle.

of pus into the nose. Some present with an orbital swelling going up and down and others with acute ruptured frontal sinus which may have broken through the skin to produce a fistula. Three mucocœles and 3 pyocœles out of a total of 37 were operated upon by the intranasal approach with apparent success.

A young lady was referred from the Eye Department in March 1958 with a one year history of proptosis forwards and laterally (Fig. 13).

An X-ray showed a large mucocœle which presented in the middle meatus. Mr. Black washed out the antra and opened the mucocœle intranasally packing it with B.I.P.P. which produced a magnificent demonstration of the extent of the mucocœle (Fig. 14). She remains well from this standpoint but she still has bronchiectasis although her sinusitis is somewhat improved.

FIG. 14.
Radiograph to show the mucocœle packed with B.I.P.P. gauze.

However, I prefer to approach a mucocœle or pyocœle by an external operation. Mrs. M. R. (Fig. 15) was admitted under the care of an ophthalmologist and neurosurgeon and because of massive proptosis a tarsorraphy was performed and 20 ccs. of pus aspirated from the orbit. Unfortunately after prolonged antibiotic therapy the eye remained displaced laterally and although an intranasal swelling could be seen, an external approach demonstrated 2 pyocœles in the anterior part of the ethmoid labyrinth and a mucocœle of the middle turbinate. None of these communicated with one another and therefore an intranasal approach would have been unsatisfactory.

I am grateful to Mr. Thomas of Carlisle for the case shown in Fig. 16 illustrating a classically extensive mucocœle. All swellings of the anterior wall are not mucocœles. Fig. 17 shows a patient with a swollen anterior wall and cystic appearances on X-ray (Figs. 18 and 19) but the sinus contained only air and an apparently normal fronto-nasal duct. What is a pneumosinus frontalis?

FIG. 15.

Mrs. M. R. Patient had had a tarsorraphy performed because of marked proptosis during the acute phase of the pyocœle.

FIG. 16.

Case of Mr. R. Thomas showing an extensive mucocœle.

FIG. 17.
Non-painful swelling of forehead.

(18)

(19)

FIGS. 18 and 19.
Radiographs showing the cystic appearances of the frontal sinus of the patient shown in Fig. 17. This was a case of pneumosinus frontalis.

Mr. R. W. presented with an orbital swelling of one month's duration which after 2 weeks had become more inflamed and tender (Fig. 20). A rubbery mass could be felt laterally and both nasal cavities contained polypi. An incision was made laterally through a very thick wall of an abscess, releasing a large quantity of pus. An X-ray of sinuses showed a

FIG. 20.
R. W. A pyocœle of the frontal sinus.

chronic osteitis of the maxilla. A bilateral combined transantral and intranasal ethmoidectomy was performed when the acute phase was over and at a later date an osteoplastic frontal flap of Bergara's type through an an incision in the eyebrow was performed. A very thick frontal mucosa was entirely removed and dura, with an overlying sequestrum, was found exposed at the lateral extension of the sinus. The sequestrum was removed and the fronto-nasal duct cleared of mucosa. The wound was closed without external drainage (Fig. 21) and within 5 weeks there was already partial obliteration of the frontal sinus. This case illustrates a staged approach to the problem which I have used with success.

At least 19 fistulae presented during the past 30 years (Table IV). They followed upon unsatisfactory operations, pyocœles, and acute orbital ruptures. As would be expected the majority appeared at the inner canthus, the only patient whose fistula communicated with the conjunctival sac died soon afterwards from an intracranial complication.

Mr. J. W. was referred by an ophthalmologist in October 1953 with

FIG. 21.

R. W. Two weeks after on obliterative frontal operation. Note the line of the incisions (a) in the lateral part of the upper lid (b) through the eyebrow.

TABLE IV.

FISTULAE—TOTAL 19.

Post-op	9 (2 had local osteitis)	
Pyocæle	6	
Ruptured Frontal Sinus	4 (2 had local osteitis (1 had osteitis with intracranial complications)	
Site of Fistulae	*Forehead*	3
	Inner Canthus	15
	Conjunctiva	1
Operations	1 Kuhnt. 17 Howarth	

pain and swelling of his left upper lid and a lot of pus in the left middle meatus. The left antrum was opaque. 600,000 units of distaquaine penicillin given twice daily and repeated antral washouts produced such a marked improvement that he was discharged home 5 days later but at the time of his discharge from the out-patient department there was still clinical evidence of sinusitis. Some 6 months later he developed a fistula above the inner canthus which gave complete relief of symptoms but produced a gross ectropion (Fig. 22) which was noticed 2 years later when he developed pain and swelling of the right upper lid. There was

a gross pansinusitis, the right frontal sinus floor was deficient, and the pyocœle was drained while the patient received penicillin and streptomycin. Two weeks later a right Caldwell Luc and external fronto-ethmoidectomy of drainage type was performed—interestingly enough the main pathology lay in the antrum and ethmoids and the frontal sinus mucosa was regarded as normal. After another week the left side was opened and the findings were similar except that the frontal sinus was full of pus although the mucosa appeared to be almost normal. He made an uninterrupted recovery and his name was placed on the plastic surgeons waiting list for correction of his ectropion. While on the waiting list for 3 years the need for a plastic surgeon's help has gone (Fig. 23).

(22) (23)

FIG. 22.
J. W. The patient has a right pyocœle and a left sided fistula with secondary ectropion.

FIG. 23.
J.W. Three years later.

The standard departmental teaching for undergraduates is that any fistula occurring above the level of medial palpebral ligament is due to a frontal sinus lesion (Fig. 24). Probing, X-rays and injection of the fistula do not add much information and frequently the findings confuse the issue.

Mrs. I. F., a lady of 50 years, had her left lacrymal sac excised for epiphora in November 1950. Three months later she developed swelling and discharge from the left upper eyelid. Although there was clinical evidence of sinusitis the radiographs of the sinuses did not suggest the presence of any lesion in the frontal or ethmoid sinuses and a lipiodol injection of the fistula failed to show a communication with these sinuses.

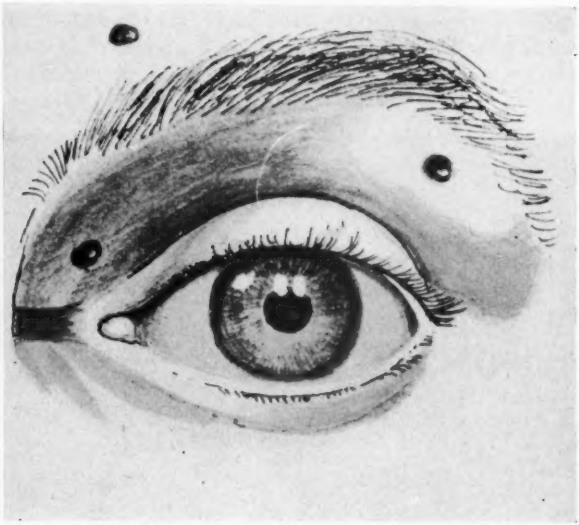

FIG. 24.

Diagram to show the common sites of fistulae. Note their relation to the medial palpebral ligament.

Although at operation the eye surgeon thought that the injected Methylene blue went into the ethmoids he drained the orbital abscess, excised and sutured the fistula edges. It broke down and 2 years later at external frontal operation there was an enormous pyocœle of the frontal and ethmoid sinuses. A drainage type of operation produced a satisfactory result except that the patient still complains of a watery eye. This case history has always emphasized to me the confusion met in over-investigating a clear-cut clinical picture. I believe that all cases of fistula should be approached externally and the prime lesion treated. In our records only one patient whose fistula persisted after several operations required obliteration of the frontal sinus by removal of the anterior wall. In several other cases the fistula was only healed when the underlying osteitis was cured.

Frontal Sinusitis and its Complications

Osteitis of the skull is frequently classified as spontaneous or post-operative, and the latter at the beginning of this century was dreaded for it was typically a rapidly spreading lesion. Only one of our cases developed post-operative osteitis in the past 30 years when there was no evidence prior to operation of osteitis as indicated by a ruptured frontal sinus. This patient, however, had gone blind as a result of sinusitis one week before nasal polypi were removed and death resulted from pyæmia due to a superior longitudinal sinus thrombosis.

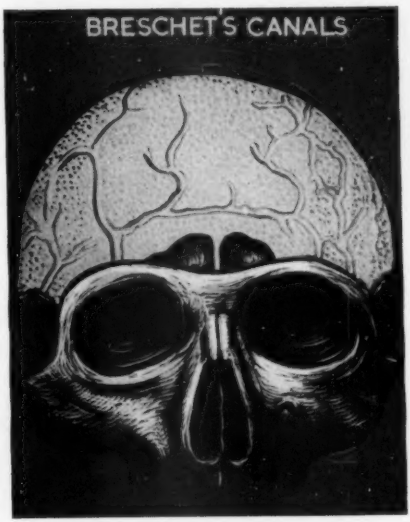

FIG. 25.
Diagram to show diploic veins—Breschet's Canals (After Skillern).

Osteitis of the skull presents in various clinical forms but it is generally agreed that the clinical patterns are no more than the different stages of the same disease in which thrombophlebitis of diploic tissue predominates. The resistance of the patient, the virulence of infection, and latterly the use and abuse of antibiotic therapy modify the thrombophlebitic lesion in bone, so producing in one a localized lesion, in another a spreading disease, or even sequestration and sometimes sclerosis. The chronic form of the disease represents a prolonged acute form. Since at onset this is a thrombophlebitic lesion of Breschet's canals in the diploe (Fig. 25) and bone destruction does not usually occur until later, then vigorous antibiotic therapy might be expected to produce complete resolution of the disease before massive bone destruction had occurred.

Pneumococci, staphylococci and streptococci were the commonest organisms found.

A 10 year old boy (Figs. 26, 27, 28 and 29) was admitted with a 13 day history beginning with a cold and fever. Seven days after onset he took to his bed and 48 hours before admission the mother noticed a swelling over the left eye which gradually increased in size. He had also vomited. Lumbar puncture showed 270 cells (52 per cent. polymorphs)

FIG. 26.
Acute osteomyelitis of the frontal bone

in the C.S.F. A nasal swab grew *Staphylococcus aureus*. Treatment by penicillin and aureomycin for 3 weeks produced resolution of the disease. (Fig. 29).

As late as 1940 Mosher would have recommended complete removal of the frontal bone, as well as the anterior and posterior walls of the frontal sinus and even then there was little more than a 50 per cent. chance of success. In some cases where maxillary sinusitis is present, antral lavage or surgical drainage may be necessary or perhaps an external drainage through the floor followed by external fronto-ethmoidectomy, particularly when fronto-nasal duct function is poor due to granulations or polyps. However, inadequate dosage of antibiotics at this stage may fail to produce resolution of the lesion, and yet such medical therapy when allied to the patient's resistance may be great enough to limit spread, and allow of local bone necrosis. The external swelling, associated with a subperiosteal abscess, is frequently limited by the suture lines.

FIG. 27.
Radiograph to show the opaque antrum in the absence of changes in the frontal bone.

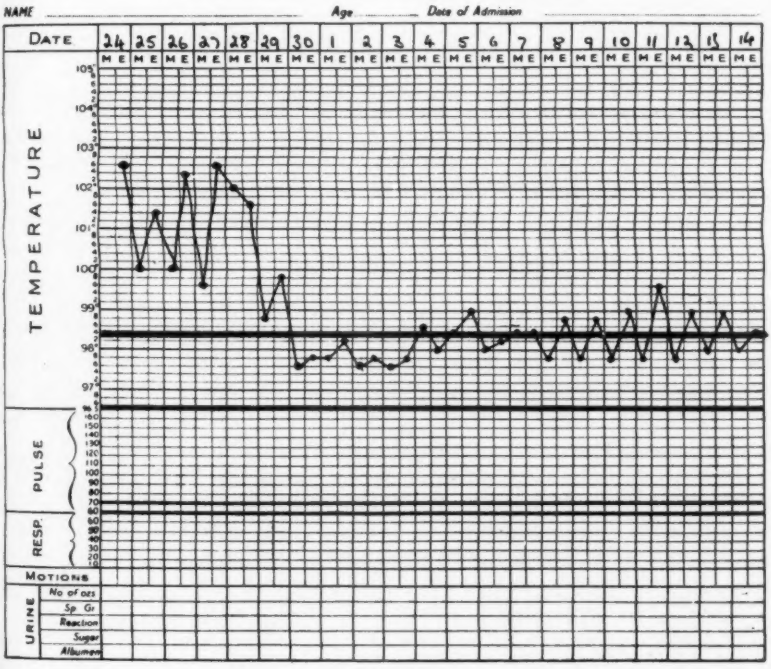

FIG. 28.
Temperature chart of patient in Fig. 26.

FIG. 29.
The same patient after three weeks antibiotic therapy.

A 48 year old man (Fig. 30) had developed pain in his eye 6 weeks previously and had had a swelling of the left upper eyelid for 2 weeks. Only small doses of aureomycin had been prescribed. On admission he had a fluctuating swelling of the brow beyond the hairline, limited only by the coronal suture. The abscess was incised and drained, and penicillin and streptomycin given. Two days later, when he had improved, an X-ray showed a well-defined sequestrum (Fig. 31). Five days after the original incision the sequestrum was removed through a wrinkle in the brow (Figs. 32 and 33). The swelling of the upper lid persisted for about one month after admission and penicillin was continued for 2 months. He remains well, without further trouble, 4 years later. No operation was directed to the very small frontal sinus which had been the cause of his trouble, since it had recovered with antibiotic therapy alone.

In my view the treatment is to drain the abscess, to give a prolonged course of antibiotics to control the osteitis and to assess with serial radiography the state of the sequestrum, and to remove it when separated, through a convenient non-deforming incision. However, if the whole anterior wall had sequestrated, deformity must follow and repair by tantalum or ilium should be deferred for some years (Figs. 34 and 35).

Chronic localized ruptures through the anterior wall do not produce ill patients and are readily treated by antibiotics, simple incision and drainage, and later assessment of the frontal and other accessory sinuses

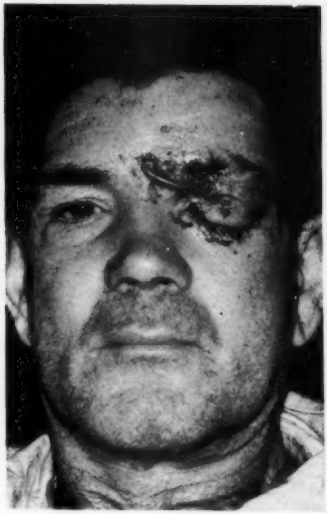

FIG. 30.
Patient after incision and drainage of the large subperiosteal abscess.

FIG. 31.
Radiograph to show the sequestrum in the frontal bone.

FIG. 32.
The sequestrum.

FIG. 33.
The patient 4 weeks later. The incision for removal of the sequestrum can still be seen.

FIG. 34.
Deformity produced by removal of the anterior wall of the frontal sinus in a case of osteitis.

FIG. 35.
Same patients as Fig. 34 after repair by ilium some years later.

as in the case of pyocœles or fistulae to determine whether an external frontal procedure is needed. However, not all small anterior swellings in a fit patient are due to a local fistula; they may be associated with chronic sclerosing osteitis of the frontal bone. The lack of symptoms may lead the patient to the wrong department.

A 76 year old patient had noticed a small swelling in the midline of the forehead for 5 weeks and it had been slightly tender for 2 weeks. A general surgeon incised this, thinking it was a neoplasm, but only found thick pus and some granulations. However, an X-ray of sinuses showed an obliterative frontal sinusitis i.e. sclerosis of the frontal bone due to osteitis (Fig. 36). Distaquaine penicillin 600,000 units and ½ gram streptomycin were given twice daily and an external frontal operation performed

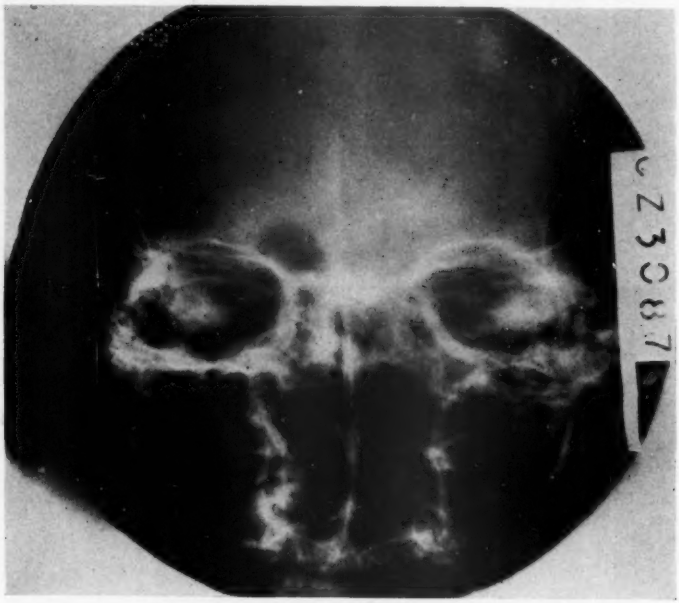

FIG. 36.
Obliterative frontal sinusitis—sclerosing osteitis of the frontal bone.

12 days later. The frontal mucosa was seen to be normal but as is frequently the case in this type of lesion the ethmoids were full of polypi. One month later, because the eyes became swollen, the drainage tubes were removed and penicillin was continued for a further 6 weeks. The patient's frontal sinus remains well but unfortunately he broke his femur soon after discharge from the department.

Spreading osteomyelitis progresses by Breschet's canals producing subperiosteal abscesses and sequestra at various points throughout the skull. Prior to the era of chemotherapy, the progress of the lesion was remorseless. I am grateful to Mr. R. Thomas for pictures illustrating

such a case (Figs. 37 and 38), wherein simple drainage of the abscesses and antibiotic therapy sufficed for complete recovery.

Osteitis, leading to sequestration or spread, may affect the posterior wall of the sinus with the production of an extradural abscess. Often, in an osteomyelitic lesion of the skull, the inner plate is mainly involved and it is not surprising that intracranial complications may arise without

FIG. 37.
Spreading osteitis of the frontal bone (Case of Mr. R. Thomas).

any external evidence of frontal sinus disease. A 22 year old man was admitted under the care of a physician with severe headache and neck stiffness following a feverish illness 8 days previously. Only 33 cells (90 per cent. polymorphs) were found in the C.S.F. He was treated with penicillin and streptomycin and discharged home as satisfactory, confidently diagnosed as a virus meningitis. On the day of his discharge a small lump appeared on his forehead. This gradually increased in size and led to re-admission to a general surgical ward. An X-ray of his frontal bone showed an osteitic area 1½ in. in size but the lateral radiograph shows that the posterior plate of the frontal base was mainly affected (Fig. 39). He recovered after excision of the anterior wall of the frontal sinus, and prolonged antibiotics. Later the deformity was repaired with tantalum.

FIG. 38.
Same case as Fig. 37 treated by incision and drainage, supplemented by penicillin. The patient made a full recovery.

FIG. 39.
Lateral radiograph of the skull showing involvement of the inner plate of the frontal bone.

Incidentally, during the search through the hospital records, a surprisingly large number of patients labelled "virus" meningitis never had the ear or nose examined by a skilled individual. Even in the case of pyococcal meningitis, if the ear was not at fault, no mention was made of the state of the nose. This is important, since patients no longer appear to die from osteomyelitis of the skull but rather of further complications, and yet it is necessary to treat the osteitis more vigorously than the meningitis—otherwise the lesion relapses. Many cases of rhinogenic meningitis clear up with antibiotic treatment and more often than not

FIG. 40.
Radiograph showing osteitis of the skull with a sequestrum. This was the first convincing radiological evidence appearing 6 months after the clinical evidence.

the causal acute sinus lesion incidentally resolves. This is not true in the case of chronic lesions which ought to be sought and treated. It is also evident that "virus meningitis" is in many cases a partly treated pyococcal meningitis without the finding of bacteria in the cerebrospinal fluid.

It is generally held that radiological evidence of osteitis of the frontal bone does not appear for 7-10 days after the clinical onset of the lesion. Antibiotics do produce a delay in the appearance of radiological evidence and in the case of Mr. J. R. the delay was as long as 6 months. This 19 year old man was admitted on the 24th June 1949 to a general physician's ward with suspected tuberculous meningitis. He gave a history of 3 weeks nasal discharge with a dull continous headache, his neck was stiff and papillœdema was present. Streptomycin was given systemically

and intrathecally for 5 months. Soon after admission the neck stiffness became worse, he had epileptiform fits, and weakness of one arm which improved within 7 days. After 2 months the papillœdema disappeared and in general he was very well apart from an odd fit. During his second week in hospital he said that he had noticed some swellings of the scalp while combing his hair 3 weeks previously. These were noted. During the next few months they increased in size and were aspirated and eventually incised. An anaerobic non-hæmolytic streptococcus was cultured, but in spite of repeated negative findings for tubercle bacilli, the diagnosis was not altered until the first radiological evidence of osteitis of the skull was noticed in December 1949 when he was transferred to the neurosurgeons for excision of the osteitic bone (Fig. 41). This is a magnificent

FIG. 41.
J.C. Acute spreading osteitis of the skull.

example not of masking of symptoms but of a total failure of the medical attendants to agree on the significance of the symptoms and signs. The fixed idea of tuberculosis reigned supreme and the prolonged delay in the appearance of X-ray changes only encouraged the error in spite of typical subperiosteal abscesses—the Pott's puffy tumours. There are great dangers in accepting pre-chemotherapeutic criteria of diagnosis in the present era of medicine.

Longitudinal sinus thrombophlebitis, as a single complication of frontal sinus disease, is very uncommon and few case-histories are found in the literature. More frequently it is associated with subdural and cerebral abscesses, and meningitis, and it may then be regarded as part of a multiple intracranial complication, although it may be the predominantly lethal lesion.

In December 1945, a 20 year old male presented at hospital with a 3 day history of pain and swelling over the right frontal bone. Sulphadiazine and 200,000 units of penicillin daily were given. He developed

FIG. 42.
J.C. Osteitis of the humerus developing nearly 6 months later and only 2 weeks after his discharge from hospital as cured.

spreading œdema of the scalp and had numerous rigors. Just before he died, he developed neck rigidity, hemiparesis and epileptiform fits. The whole history from beginning to end covered 7 days and autopsy revealed subperiosteal, extradural and subdural abscesses together with longitudinal sinus thrombosis. The dosage of chemotherapeutic agents at that time was insufficient to control the overwhelming infection in which pyæmia predominated. Today with more efficient use of antibiotic therapy recovery would be expected. Occasionally, a patient may recover from osteitis of the skull complicating frontal sinusitis and yet develop a pyæmic abscess shortly afterwards. J. C. (Fig. 41) is such an example, for after recovering from acute spreading osteitis of the skull which lasted 5 months, he developed osteitis of the humerus 2 weeks after discharge from hospital (Fig. 42). I believe this to be an example of superior

longitudinal sinus thrombosis incompletely controlled because sulphonamide therapy was not sufficiently potent. He eventually recovered.

Cavernous sinus thrombosis is usually considered as a complication of frontal sinusitis. However, I have not found one convincing example in about 50 cases which have occurred in the Infirmary records.

Mrs. A. W., a 61 year old lady, developed a furuncle in the right side of the nose 7 days before the pictures shown in Figs. 43 and 44 were taken. She became blind in the right eye as a result of optic atrophy. Antibiotics sufficed to cure the lesion.

FIG. 43.

Cavernous sinus thrombosis. This followed a furuncle in the nasal vestibule. Some of the thrombosed vessels in the forehead can be recognized.

Other causes of cavernous sinus thrombosis, apart from skin sepsis, have been otitis media complicated by superior petrosal and lateral sinus thrombosis, and quinsies (Fig. 45). Spread from a quinsy to the cavernous sinus either extends upwards along the internal jugular vein or through the pharyngeal venous plexus. However, whatever the cause, a gangrenous sinusitis was frequently found at autopsy and appears to have been considered of ætiological importance in the past. Clearly, this is no more than an extension of retrograde infective thrombophlebitis to paranasal sinus mucosa from the cavernous sinus and it may be regarded as a result rather than a cause of cavernous sinus thrombosis.

Cerebral thrombophlebitis, although a frequent extension of intracranial sinus thrombosis, may arise independently as a direct result of spreading thrombophlebitis from the dural and diploic veins across the

FIG. 44.
Lateral view of the patient in Fig. 43.

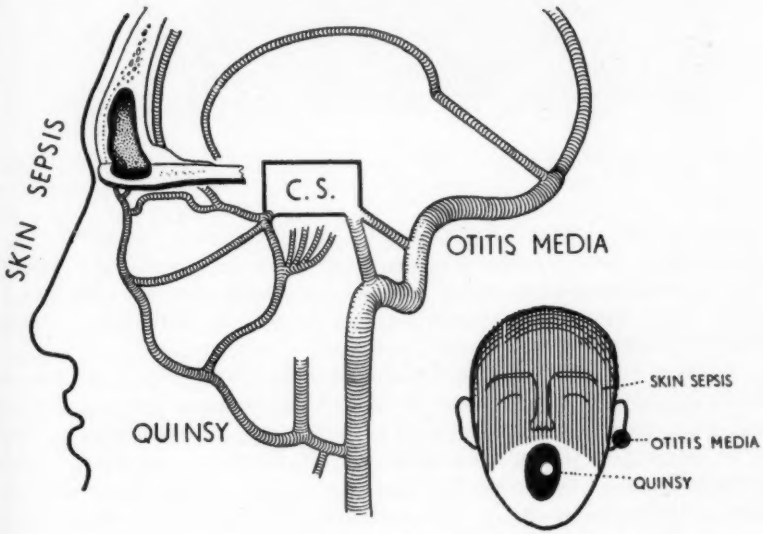

FIG. 45.
Diagram to illustrate the common causes of cavernous sinus thromophlebitis

meningeal space to cerebral tissue. It is also a pathological state which precedes the formation of a cerebral abscess. Now that vigorous antibiotic treatment is administered in the early stages of fulminating disease, cerebral abscess formation may be arrested at the thrombophlebitic stage and total resolution of the disease attained by therapy alone. Although cerebral thrombophlebitis was an uncommon diagnosis in the past, it is likely to be a more frequent finding at the present time

FIG. 46.

Osteitis of the skull with a small sequestrum. This patient later developed a cerebral thrombo-phlebitis.

because of the modified natural history of brain abscess (McNally 1954). When small doses of antibiotics are given as treatment for ruptured frontal sinus or osteitis of the frontal bone, they may be sufficient to protect the brain against abscess formation and yet allow cerebral thrombophlebitis to develop.

In June 1950 a 41 year old man was seen, giving a 4 weeks history of swelling of the right forehead and eye following a prolonged nasal discharge. 400,000 units of Procaine penicillin were given daily for 10 days as an out-patient (too small a dose) and although the swelling subsided, a residual mass remained. Ten days later the swelling recurred, and this no longer responded to the out-patient dosage of penicillin. When admitted the abscess had already burst. The radiograph taken showed osteitis with a sequestrum (Fig. 46), and he was also noticed to have bilateral nasal polypi. In July 1950 a Howarth type of drainage operation

was performed with skin grafting of the fronto-nasal duct. He was re-admitted again in August 1950 because of recurrence of frontal pain, dragging of the right foot and weakness of the arm and aphasia. The neuro-surgeons found evidence of encephalitis of the temporosphenoidal lobe. He recovered.

This is a striking example of ineffectual treatment modifying the course of an osteitis. Antibiotics were never given in proper dosage for a long enough period and the hemiplegia was a result of cerebral thrombophlebitis well removed from the original source of the infection. I do not quote this example as a case of thrombophlebitis preceding a cerebral abscess but rather as an example of poor management.

Table V shows that in our case of ruptured frontal sinus approximately one in four have or will develop osteitis of the skull and one in five or six may develop an intracranial complication. The incidence has

TABLE V.

RUPTURED FRONTAL SINUSES.

	1930-37	1938-44 (Excluding 1942)	1945-59	Total
R.F.S.	47	49	74	170
Osteitis	10	13 (1 tuberculous)	21	44 (26%)
Extradural Abscess only	2	3	4	9
Intracranial Complications ..	6	7	10	23 (13·5%)

not changed much in the past 30 years. However, if we look more closely at osteomyelitis of the skull, there has been an enormous increase in the cure rate of the acute lesions as a result of more efficient antibiotic therapy (Table VI). Patients do not die of osteitis itself but of its complications. In fact, all deaths arising initially from frontal sinusitis are due to the intracranial complications. Table VII indicates that the persistent killer is a brain abscess, often associated with other intracranial lesions, but since 1947 less frequently associated with osteitis or ruptured frontal sinus. The overwhelming acute suppurative intracranial lesions, such as meningitis or intracranial sinus thrombophlebitis are now controlled by antibiotic therapy, a fact better demonstrated in the very much commoner otogenic intracranial suppurations. Of the 3 doubtful cases mentioned 1 died and 2 recovered. Thirteen cases of frontal lobe abscess have been seen in the past 30 years and only 4 recovered all of which had become encapsulated. The one recovering in 1935 had a 5 week history of vomiting, drowsiness and slow cerebration, and showed osteitis of the forehead. An external frontal operation with

TABLE VI.

OSTEITIS OF SKULL.

1930-37	10 cases	6 deaths	1 anæsthetic 5 intracranial complications all localized lesions
		4 recoveries	1 had sequestrum of post. wall. 1 frontal lobe abscess
1938-44 (Sulphonamides)	12 cases	4 deaths	all acute lesions with intracranial complications
		8 recoveries	4 acute lesions 4 localized lesions
1945-59 (Antibiotics)	22 cases	2 deaths	1 spreading 1 localized—both intracranial complications
		20 recoveries	4 acute with antibiotics alone 1 spreading 4 acute with sequestration 1 chronic localized 1 obliterative

TABLE VII.

INTRACRANIAL COMPLICATIONS. TOTAL 33—23 PRESENTED AS R.F.S.

1930-1937	8 intracranial complications (6 had osteitis)	6 deaths	4 Multiple intracranial with cerebral abscess. 1 meningitis, long sinus thrombosis and pyæmia 1 meningitis as result of surgical trauma
		2 recoveries	1 frontal lobe abscess 1 extradural abscess
1938-1944	8 intracranial complications (6 had osteitis)	5 deaths	1 cerebral abscess with subdural 1 cerebral abscess 2 meningitis 1 sup. long. thrombosis with pyæmia
		3 recoveries	2 extradural abscesses 1 encephalitis
1945-1959	17 intracranial complications (10 had osteitis)	5 deaths	4 brain abscesses 1 meningitis (moribund on admission)
		9 recoveries	4 extradurals 1 encephalitis 3 meningitis 1 brain abscess
		3 doubtfuls	Cerebral abscess
1947-	Intracranials with ruptured frontal sinus 2—both had osteitis. Intracranials without ruptured frontal sinus 7—1 had osteitis.		

removal of a large sequestrum of the posterior wall released pus under pressure from an extradural abscess and the communicating frontal lobe abscess. Another patient survived long enough to have his frontal lobe excised, having had a 2 months history of osteitis of the skull. A fistula through the posterior wall found at external frontal operation

communicated with an extradural abscess. The patient's only symptoms were headaches and vomiting, and later papillœdema was found.

Papillœdema has not been present in the acute cases since in cerebral abscess it takes at least 2-3 weeks to develop, and it cannot be regarded as a useful diagnostic sign. When present it gives an indication that the abscess is one of some duration. We must admit that often the diagnosis was reached when the patient had become moribund.

In 1951 a 17 year old man developed frontal sinusitis following influenza from which he seemed to recover. Ten days later he complained

FIG. 47.
Abscess in the frontal lobes. This patient died from supratentorial hydrocephalus.

of headache and fainted, and then vomited. He was kept under observation in hospital under a physician for 2 days without being diagnosed. His headache continued and he occasionally vomited. Two weeks later he had a temporary disturbance of vision and became drowsy. On admission the right pupil was dilated but his reflexes were regarded as normal. The C.S.F. was under increased pressure and contained 100 leucocytes. He died 3 days after admission undiagnosed. Necropsy revealed a cerebral abscess with gross supratentorial hydrocephalus due to uncal herniation through the opening of the tentorium cerebelli (Fig. 47).

Another common cause of death is rupture into the ventricle. A 22 year old man developed headache after a bad cold and this became more generalized within a week. He soon began to vomit and 12 days after the onset of the cold he was admitted to hospital with a temperature of 101° and

neck stiffness. The C.S.F. contained 2,000 cells (95 per cent. lymphocytes). A few hours later his breathing became stertorous and cyanosis developed before respiration ceased. Artificial respiration and frontal burr holes with drainage of the abscess were carried out without benefit to the patient who had perhaps already succumbed from rupture of the abscess into the ventricle. These sad tales were surprisingly common. In retrospect, every case should have been suspected on clinical grounds, even if not diagnosed in detail. No neurosurgeon can be expected to cure these acute cases if they never reach him. Acute brain abscesses present in ear, nose and throat departments accompanying frontal sinus disease. It is our duty to spot these unusual cases, to exert every effort to reach a firm diagnosis, and seek the help of our colleagues. The frontal lobe is a silent area in the cortex and the absence of localizing physical signs makes diagnosis difficult. An acute abscess in the frontal lobe is more likely to rupture into the anterior horn of the ventricle before it produces signs of muscle weakness and indeed it is only the large chronic abscess which becomes a space-occupying lesion, similar to a neoplasm, which may produce muscle paresis by virtue of the abscess itself. This never occurred in any of our cerebral abscesses and it is evident that such paresis is more frequently due to an abscess readily spreading through the subdural space, usually over the medial and inferior surfaces of the frontal lobe. In Fig. 48 I show the lateral surface merely to reinforce the argument. Several features have forcibly struck me in reading through some hundreds of case-histories. Early intracranial hypertension is manifested by headache and vomiting, and not by papillœdema. Vomiting in particular does not occur in frontal sinusitis or osteitis of the skull, and when present we *must* diagnose an intracranial complication. Mrs. Brown who was 6 months pregnant, had a pansinusitis following a cold and developed a "ruptured and frontal sinus". She was treated with aureomycin and, although the orbital swelling became less in 24-48 hours, the patient began to vomit. She was immediately transferred to hospital and even in the absence of neck rigidity a lumbar puncture showed 330 cells (mainly polymorphs) in the C.S.F. She was an early case of meningitis and recovered rapidly with vigorous antibiotic therapy (Fig. 49).

I have tried to summarize in Table VIII the main diagnostic features of intracranial complications.

Headache may frequently change its character from a local pain to a diffuse headache—this frequently means an extension beyond the sinus and all aids to diagnosis should be brought to bear.

One rigor or a high swinging temperature and fast pulse indicate a pyæmia. At the time when treatment is most effective against pyæmia there are no physical signs and blood culture in my experience is sterile. Small doses of antibiotics may slow up the course of pyæmia and

SPREAD OF INFECTION.

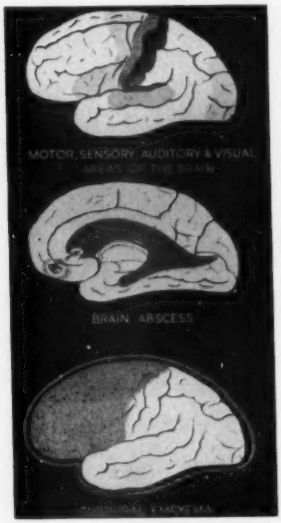

FIG. 48.

Diagram to illustrate the absence of physical signs in a frontal lobe abscess and their presence in a subdural abscess.—See text.

TABLE VIII.

SYMPTOMS AND SIGNS OF INTRACRANIAL PYOGENIC SUPPURATION COMPLICATING A FRONTAL LOBE ABSCESS.

1. Patient is UNWELL.
2. Headache } At least an extradural abscess—may be much more.
3. Vomiting. }
4. Rigors. Sup. Long. Sinus
5. Epileptiform Fits. Subdural abscess
6. Drowsiness and Coma. { Cerebral Thrombophlebitis / Meningitis / Cerebral Abscess
7. Neck Rigidity. Meningitis (Sometimes late in onset)
8. Temperature and Pulse Charts.
   Physical signs absent in early stages

confuse the issue, but one rigor should be regarded as diagnostic of pyæmia and vigorous therapy started.

Epileptiform fits have not occurred in any of these cases where there was no evidence in the surviving or dead patient of a subdural abscess. In the early days of such a lesion, complicating an acute frontal sinus disease, vigorous antibiotic therapy alone may give control and produce a satisfactory result, but if it fails to control the epileptic attacks or if

they are of late onset, then a neurosurgical approach is necessary. Once an epileptiform fit has occurred, then the patient should be managed in close co-operation with the neurosurgeon.

Drowsiness and later coma are due to involvement of cerebral tissue. Every cerebral abscess is preceded by a cerebral thrombophlebitis

FIG. 49.
This photograph shows the patient soon after admission. She had meningitis.

of at least 2-3 days duration and if vigorous treatment is begun in this period an abscess may never develop. Proper treatment can only be started if an intracranial lesion is suspected.

Most patients with acute ruptured frontal sinusitis are not ill. If they are ill, one must assume that an intracranial complication is present.

Vigorous treatment and planning of procedure must be determined at the time the patient is first seen with a ruptured frontal sinusitis and if there is any suspicion of intracranial involvement, treatment and investigations must go along side by side. Electroencephalography and neuroradiology are of great help in reaching a diagnosis, and to establish a firm diagnosis burr holes may be needed and only full co-operation between departments is likely to produce the best results. It is interesting that of the last 125 cases of cerebral abscess examined by Dr. Gryspeerdt, our neuroradiologist, only one was a case of frontal lobe abscess and this was not a complication of sinusitis. I show the radiographs because of interest (Figs. 50 and 51). The angiogram shows displacement of the

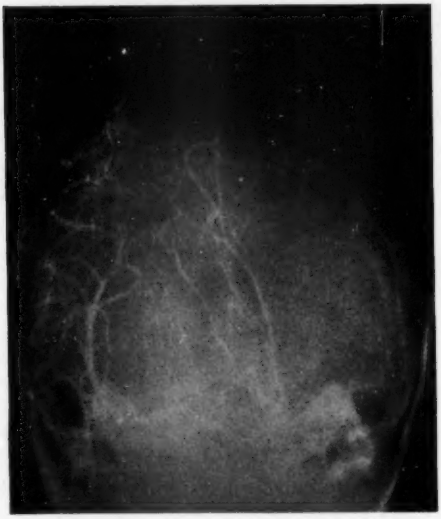

FIG. 50.
Cerebral angiogram showing displacement of the anterior cerebral artery and the absence of filling in the region of the abscess.

FIG. 51.
The same case as Fig. 50. The abscess cavity filled with thorotrast.

cerebral vessels and in the next slide thorotrast is shown in the abscess. Presumably the other frontal lobe abscesses required more urgent surgery.

I would like to thank my colleagues and teachers, Mr. Francis McGuckin and Mr. Munro Black for not only allowing me to review patients admitted under their care but also for tolerating my criticism over the years. My only hope is that this contribution has been of some value and that some of the clinical lessons learnt will help to guide others towards a more careful assessment of patients suffering from frontal sinusitis and in particular its intracranial complications. If an intracranial lesion is suspected on clinical grounds by an accurate observer, it is usually present even if accessory aids to diagnosis do not provide confirmatory information in the first instance.

Acknowledgements

I am greatly indebted to Mr. F. McGuckin for revising the script for publication. Mr. D. P. Hammersley of the Department of Surgery prepared all the original diagrams in beautiful colour and kindly redrew them in black and white for publication. The photographs and radiographs were prepared by the departments of photography of the University of Durham and the Royal Victoria Infirmary. The paper itself could never have been read nor published without the numerous drafts typed by the department's secretary, Miss D. Forster.

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HISTOLOGICAL CHANGES IN THE INNER EAR RESULTING FROM THE APPLICATION OF ULTRASONIC ENERGY

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In 1952 Krejci demonstrated that in guinea-pigs he could abolish the responses of the semicircular canals, without affecting the hearing, by irradiation with a narrow ultrasonic beam. Following irradiation, the vestibular reactions were tested by rotation and the after response measured by electronystagmography. The electrical response of the cochlea, was measured by the method of Davis.

The frequency of the ultrasound was 800 kilocycles per second and the intensity 4 watts/sq.cm. This was applied for a duration of one to two minutes and after three to four such applications the resulting nystagmus which was directed towards the ear undergoing treatment could no longer be produced. After the cochlear and vestibular response had been measured, histological examination of the labyrinth was carried out. The only change seen was hyperæmia and some extravasation of blood in the perilymphatic space.

In the light of these findings, Krejci treated one case of Ménière's disease by direct application of ultrasound to the lateral semicircular canal after surgical exposure through the mastoid cavity. This resulted in control of the vertigo, improvement in hearing and reduction of the tinnitus, and paved the way for Arslan to develop the method, which is becoming increasingly used in many parts of the world. Arslan uses a frequency of about 800 kc./s at intensities up to 10 watts/sq.cm. The duration of the application is controlled by observation of the reactions of the eyes. Irradiation is continued until nystagmus, which is first seen directed towards the treated ear, becomes reversed and is directed away from the treated ear showing that there has been a reduction of vestibular function on that side.

The effect on the labyrinth using this method has been described by de Stefani. He applied ultrasound at a frequency of 800 kc/s. to the semicircular canals of dogs at an intensity 10 watts/sq. cm. for 15 minutes. The dogs were killed for histological examinations at 8, 11 and 14 days after irradiation.

At 8 days degeneration of the neuro-sensory epithelium of the crista

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ampullaris with vacuolation and pyknosis of the nuclei was seen. At 14 days this degeneration was very marked and there was œdema of the cupula. These findings of progressive degeneration are in keeping with the clinical findings of Lumsden who attributed the occasional occurrence of modified attacks of vertigo during the first few weeks after operation to a progressive degenerative process rather than an immediate totally destructive one. Ariagno (1960) using the same type of application has applied ultrasound to the semicircular canals of monkeys. The frequency was 800 kc/s. Using an intensity of 6-8 watts/sq. cm. the neuro-epithelium of the crista showed œdema and pyknosis of the cell nuclei one week after irradiation. Three weeks after irradiation at 8 watts/sq. cm. the epithelium became detached and vacuolation and pyknosis was present. There was also degeneration of the cupula. Six weeks after irradiation with an intensity of 9 watts/sq. cm. there was advanced degeneration and fragmentation of the epithelium with further degeneration of the cupula.

Portmann has described the effects of irradiation of the labyrinth by an alternative route. The external auditory meatus was filled with fluid and used as a coupling medium between a transducer and the tympanic membrane so that ultrasound reached the labyrinth *viâ* the ossicular chain. The frequency used was 1,000 kc/s. At an intensity of 2-3 watts/sq. cm. lesions of the organ of Corti and the auditory nerve were seen after several days. At 8 watts/sq. cm. destruction of the membranous labyrinth occurred.

It seems likely that in the method of Portmann more energy will be lost in transmission across the middle ear than in the method of Arslan where there is direct application to the labyrinth. However, the results described show that by Portmann's method, a greater degree of destruction occurs in the labyrinth with the same intensity of applied ultrasound.

More recently Brain, Colman, Lumsden and Ogilvie (1960) have described their experimental work on the ultrasonic irradiation of the internal ear of cats. The intensity of the vibrations was progressively increased to reach 8 watts/sq. cm. at the end of five minutes and continued at this level for a further period of five minutes. In a series of histological examinations the earliest changes noted were vasodilatation and increased capillary permeability at the end of one half hour. After 48 hours there were severe degenerative changes in the neuro-epithelium of the labyrinth, most marked in the cristae of the semicircular canals, but occurring to some degree in the organ of Corti. After ten days there were further changes in the neuro-epithelium, but in a number of the sections the cupula did not appear to be seriously altered. After twenty days there was almost complete loss of sensory epithelium and destruction of hair cells, but again the cupula was seen to be more or less intact in some of the sections.

One of the technical difficulties of this work is the measurement of the actual intensity of ultrasound emitted by a transducer under the working conditions, and this difficulty is stressed by Lumsden and his co-workers, who suggest that the actual intensity output during their experiments was lower than the stated figures. For the experimental work at the Institute of Laryngology and Otology the following apparatus and method of calibration have been used.

Apparatus

The ultrasonic energy is generated by a highpower, 1,350 kc/s. oscillator and transducer designed and built at the Institute of Laryngology and Otology for the main part by E. B. Neergaard of Copenhagen. The original transducer consisted of a barium titanate disc working into a water coupling medium in which the ultrasound was focused by a parabolic reflector. The water system was difficult to use and was finally discarded in favour of a solid aluminium system which has a number of mechanical advantages, but in which the efficiency is considerably reduced.

The principal disadvantage in a low efficiency system is the amount of heat which is generated in the applicator, the necessary increase in drive power being of little importance as the power requirements are relatively small. (The total ultrasound output of the tip of the Italian Federici applicator, if the intensity were 12 watts/sq. cm. would be approximately 2·4 watts.)

A water cooling system in good thermal contact with the applicator head suffices to keep the temperature at a reasonable level.

Calibration

Measurements of the intensity of ultrasound output are difficult to make and it has not yet been possible to do so whilst clinical application is in progress. A system has been set up which allows the measurement of total power output into a standard load, and it is thus possible to compare the output of applicators of different design. If an accurate assessment is ever made of the loading characteristics of the inner ear, it would then be possible to calculate the actual power or intensity from such measurement. The measurement is made by delivering the ultrasound into a small absorber in which all the sound energy is converted into heat. The temperature of this absorber is held constant by a built-in electrical thermostat and the electrical heating power used by the thermostat is recorded. When the ultrasound is switched on the heat generated reduces the amount required from the thermostat. This reduction in the power supplied by the thermostat is equal to the total sound power input to the absorbing load. It should be realized that this will vary with the acoustic

properties of the absorber used. In the experiments carried out in the laboratories of this Institute plasticine has been used as being the most convenient material with which to work.

Power measurements made with this apparatus show that an intensity of 0·9 watts/sq. cm. was recorded during these experiments, and this represented a total power of approximately 0·3 watts from the applicator used.

In the present communication ultrasound has been applied to the left superior vertical semi-circular canals of three guinea pigs, the right ears being untouched in any way. The animals were killed after varying periods and serial sections were made of the whole of each temporal bone in all three animals, so that the right ears act as controls in each case.

Method

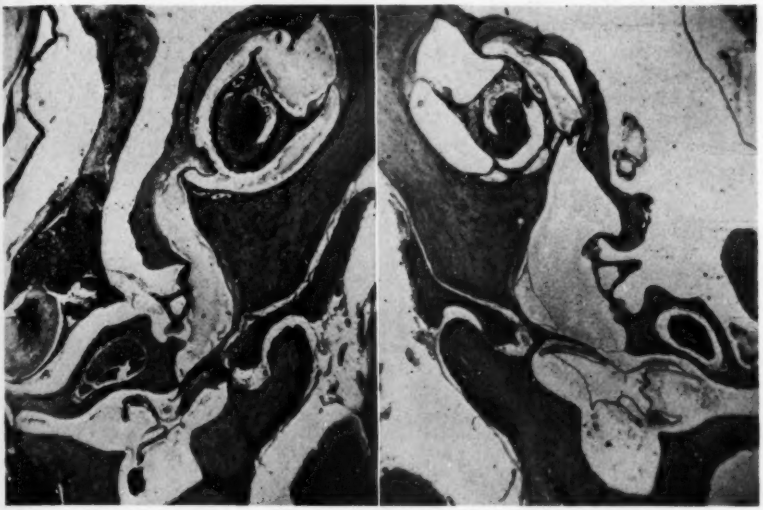
Under intraperitoneal urethane anæsthesia the superior recess of the guinea-pig was opened by a supra-auricular incision. The recess was enlarged sufficiently to allow the ¼ in. diameter face of the transducer to be placed on the superior vertical semicircular canal, without any other parts coming in contact with the transducer. A drop of saline which had been de-aerated by boiling was placed between the canal and the transducer face as a coupling medium. Interference of this coupling by blood was prevented by suction applied in the adjacent cavity. Three guinea-pigs were irradiated in this way. The first was irradiated for thirty minutes at an intensity of 0·9 watts/sq. cm. The animal was killed immediately after irradiation. In the second guinea pig, the same intensity of ultrasound was again applied for thirty minutes, but the animal was allowed to recover. It was killed seven days after irradiation. In the third animal, ultrasound at the same intensity was applied for fifteen minutes. It was allowed to recover but died on the fourth day after irradiation.

In the guinea-pig, which was subjected to 30 minutes exposure and which was killed immediately after exposure no changes were detected in any part of the internal ear. In the case of the animals allowed to recover and survive some days, considerable changes are seen to have occurred.

As can be shown by clinical observation during the exposure of the canal to ultrasound there is an immediate reduction of activity of the endorgan and thus there must be some biological change. This is not obvious histologically but becomes so after a few days. A comparable phenomenon occurs when the central nervous system is subjected to ultrasonic irradiation—functional change, but no immediate histological signs. The results of the ultrasonic application become obvious in a few days (Fry, 1954).

The following plates demonstrate changes that occurred in the internal ears of the guinea-pigs treated for thirty minutes and for fifteen minutes

respectively. In each plate the corresponding sections from the treated left ear and of the untreated right ear are presented together for comparison and the more important changes are indicated.



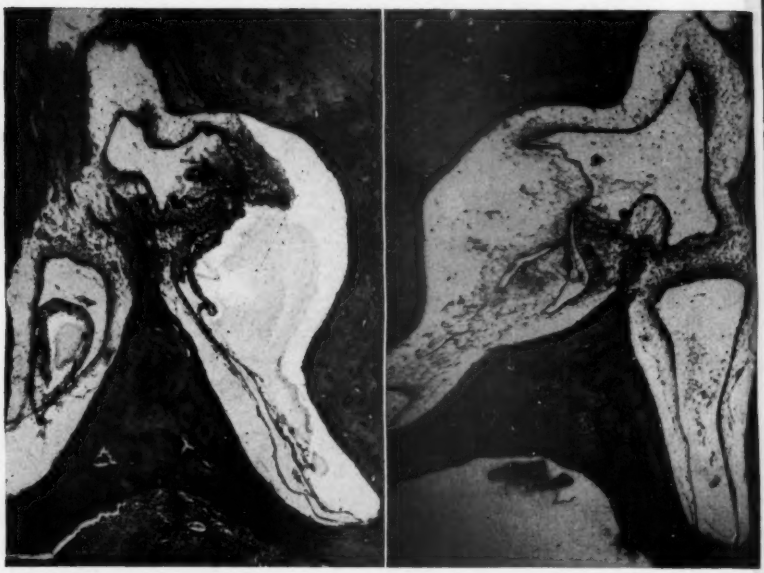
Treated Left ear × 12. Untreated Right ear × 12.

FIG. 1.

Guinea-pig No. 19 subjected to ultrasound for 30 minutes. Lived 7 days. Section through cochlea, vestibule and ampullae of lateral and superior vertical canals. Notable feature is collapse of membranous labyrinth seen throughout. The scala media does not exist as a space, the remains of Reissner's membrane lying in contact with the stria vascularis, and with the tectorial membrane. The saccule and the utricle are equally collapsed without a very marked change in the sensory epithelium of the macula of the saccule. There appears to be more degeneration of the sensory cells of the utricular macula. In the ampullae there is almost complete collapse of the endolymph sac—the sensory epithelium of the cristae being considerably degenerated. In the semicircular canals there is almost total collapse of the membranous canal, particularly in the vertical one. There is also some precipitation and a little hæmorrhage in the perilymph spaces. There are some degenerative changes in the fibres of the facial nerve.

Discussion

The effects of the application of ultrasound to the labyrinth of the guinea-pig vary according to the duration of the application and also to the time which elapses between the application and the death of the animal. In an animal killed immediately after the exposure no changes can be detected histologically. In a guinea-pig subjected to fifteen minutes exposure and surviving four days, there is some dilatation of the membranous labyrinth, and there are already degenerative processes in the sensory epithelium covering the cristae of the canals and the macula



Treated Left ear × 40. Untreated Right ear × 40.

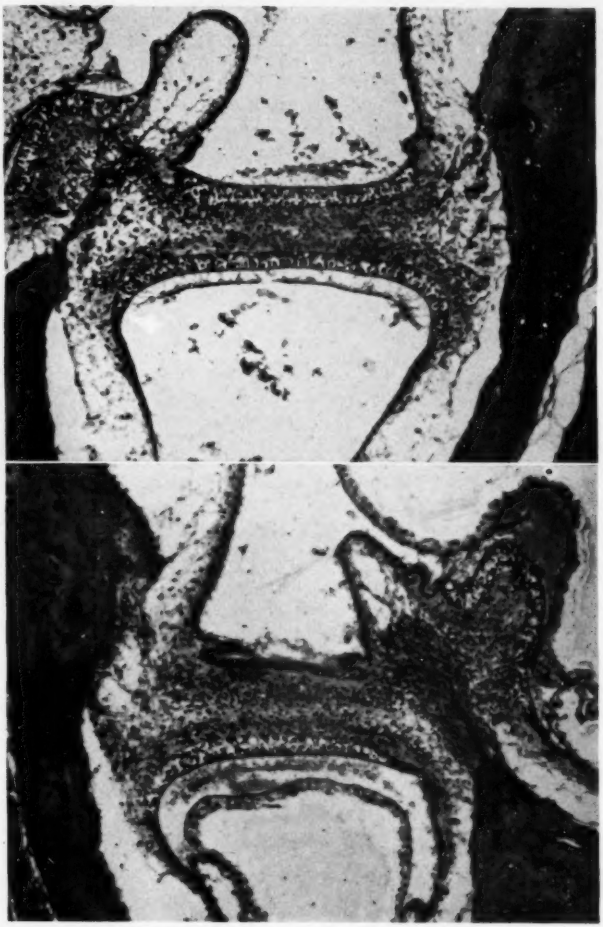
FIG. 2.

Same guinea-pig as Fig. 1. Utricle, ampullae and canals show marked collapse of endolymph sac with precipitation in endolymph and also in perilymph. Sensory epithelium of utricle appears to be entirely disorganized. Superior vertical canal is completely collapsed and there are marked changes in the sensory epithelium of the crista in the ampulla of this canal. This is more marked on the ampullary side of the crista than on the canal side. The lateral canal is not so completely collapsed and the sensory epithelium is not so degenerated as in the vertical one.

of the utricle. There are also some hæmorrhages into the region of the canals.

In an animal exposed to the same degree of ultrasound for thirty minutes and surviving for seven days there are more marked changes. The membranous labyrinth is largely collapsed. The utricle and the superior vertical canal are completely collapsed, the lateral canal somewhat less so. The scala media is empty and the remains of the ruptured membrane of Reissner lie on the organ of Corti and on the stria vascularis. The sensory epithelium is everywhere severely damaged—in the vestibule, near to the source of ultrasound there is marked vacuolation and degeneration but in the cochlea the sensory epithelium is disorganized rather than degenerated. There is however considerable atrophic change in the stria vascularis and in the connective tissue which lies beneath the secretory epithelium.

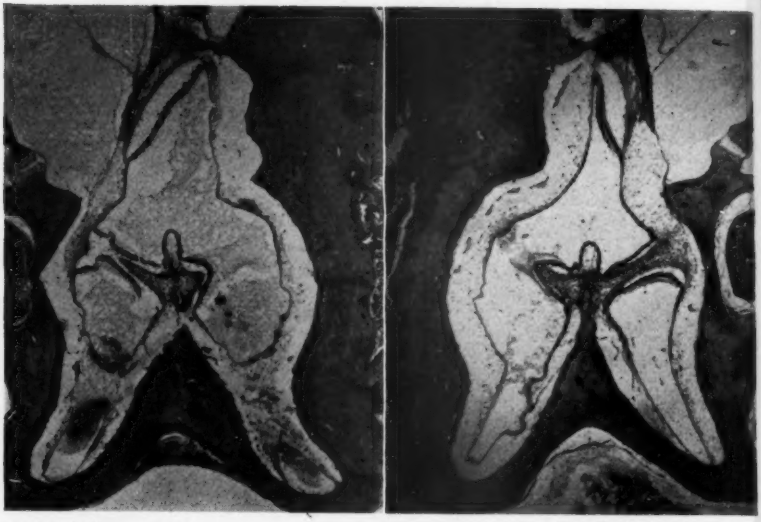
Care was taken in these experiments to direct the beam of ultrasonic



Above: Untreated Right ear × 120. Below: Treated Left ear × 120.

FIG. 3.

Same guinea-pig (No. 19) as in Figs. 1 and 2. Higher magnification of ampullary end of lateral canal. On the treated side there are seen considerable changes in the sensory epithelium of the crista. On the ampullary side there is almost complete degeneration of the epithelium with some disorganized remains of the cupula. On the canal side there is vacuolation of the sensory epithelium with exudate into the edge of the cupula and subcupular space. In the section there can also be seen a layer of secretory type of epithelium probably resulting from the collapse of the membranous canal. In the crista of the vertical canal ampulla there is also vacuolation of the sensory epithelium and a condensation of the cupula. In this region the collapse of the membranous canal is more complete.



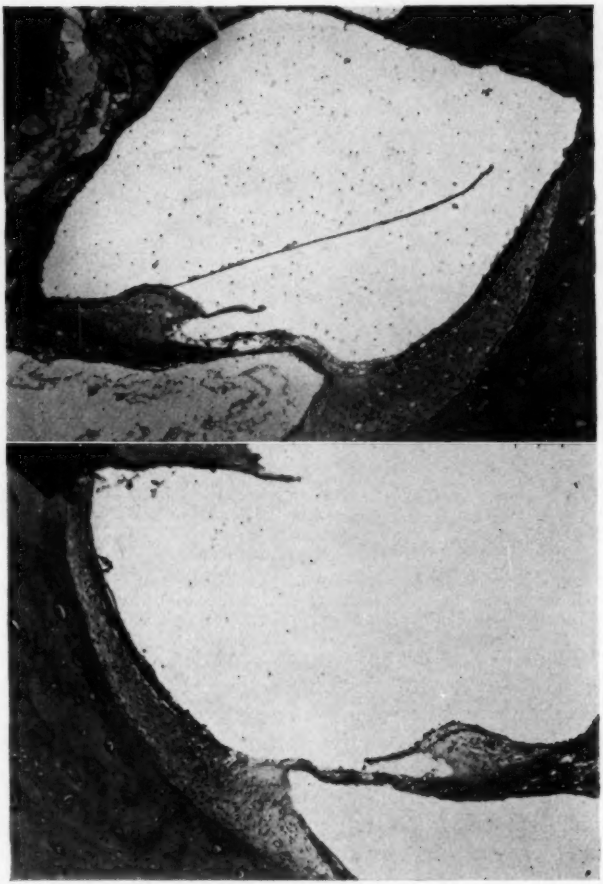
Treated Left ear × 35. Untreated Right ear × 35.

FIG. 4.

Guinea-pig No. 32 submitted to ultrasound for 15 minutes. Lived 5 days. The ampullae of the lateral and vertical canals with a portion of the canals, and the ventricles. On the untreated side there is an almost complete cupula on the crista of the vertical canal. On the treated side the cupula of the corresponding ampulla is also apparently undamaged. There is however hæmorrhage in both ampullae and to a greater extent in both lateral and superior semicicular canals. The ampullae and possibly the canals seem to be dilated rather than collapsed. In the utricle the sensory epithelium is somewhat degenerated and the otolith membrane has disappeared. The delayed changes, after 15 minutes application of ultrasound, suggest irritation of the structures of the inner ear—with probable loss of function, but in the case of the utricle not definite permanent damage.

vibration towards the ampullae of the canals but it is difficult to construct a practical applicator small enough to cover only the ampullary region, and yet emit the required intensity of ultrasound. In the human subject it is possible to do this, to destroy the function of the vestibular labyrinth, and at the same time to preserve that of the cochlea. In the guinea-pig the destruction is most marked in the vestibule but is severe enough in the cochlea to abolish function. It is obvious that the intensity of vibrations must diminish the further they travel from their source, and it appears that the force which has caused so much damage in the vestibule, has undergone reduction and causes less severe change in the cochlea. That such change has however taken place may be due to conduction of the very high frequency vibrations along the column of labyrinthine fluids to the cochlea.

It is necessary to consider how these degenerative changes are brought about. They consist in the main of collapse of the membranous labyrinth,

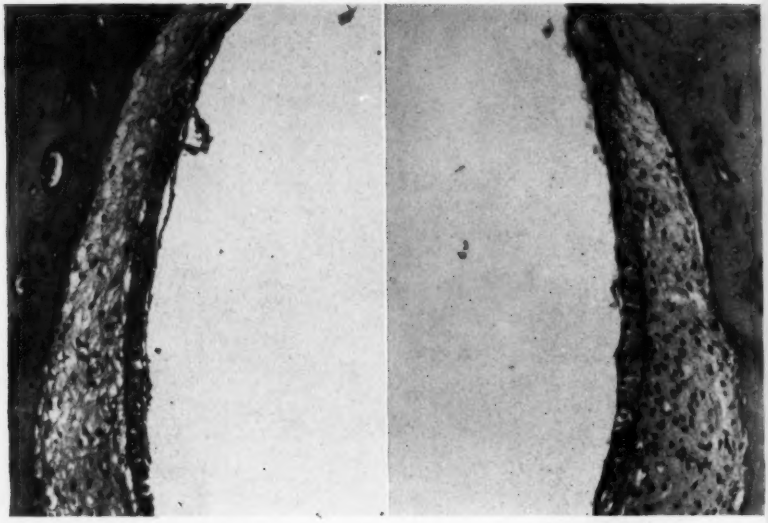


Above: Untreated Right ear × 90.
Below: Treated Left ear × 90.

FIG. 5.

The cochlea in the guinea-pig No. 19 subjected to 30 minutes ultrasound. The scala media is non-existent as a separate space. The remains of Reissner's membrane can be seen lying closely applied to the stria vascularis, and along the upper surface of the tectorial membrane and limbus.

and of vacuolation, pyknosis and atrophy of the cells of the sensory and of the secretory epithelium. As there are no changes visible histologically when an animal is killed immediately after the application of ultrasound it does not appear that the changes, which are to be observed at later periods, are due to the direct effect of the vibrations. If this were so



Treated Left ear × 240. Untreated Right ear × 240.

FIG. 6.

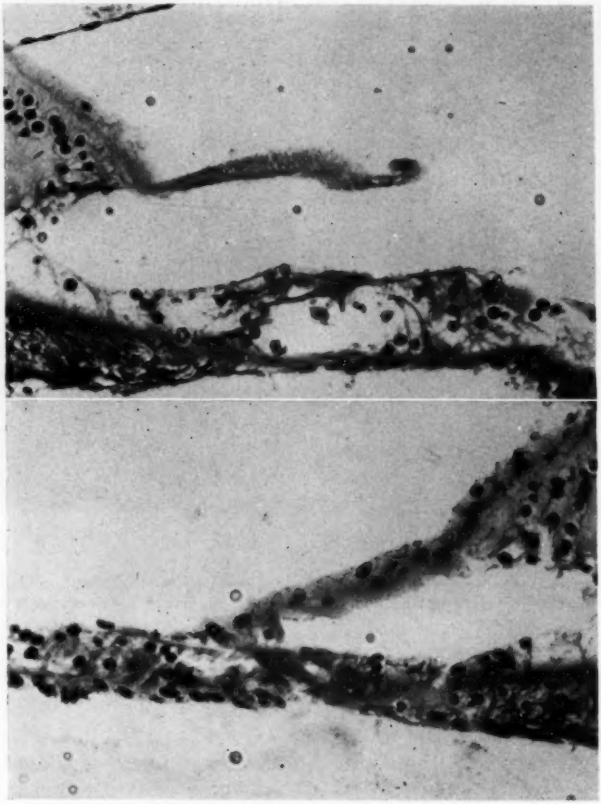
The cells of the stria vascularis are shrunken and opaque and apparently degenerated. There is notable degeneration in the subepithelial tissue deep to the stria vascularis. The remains of Reissner's membrane are seen applied to the surface of the stria vascularis.

there would be evidence of it, either in the sensory cells or in the membrane dividing the endolymph from the perilymph.

In the human subject, function is lost after a therapeutic exposure to ultrasound which is, proportionately to the size of the animal, no greater than that applied to the guinea-pig and the time of exposure is about the same. It has not been possible to study the changes which take place in the human labyrinth but by comparing the functional results in man with the histological changes seen in experimental animals, it would appear that some biological or chemical change, which interferes with function, takes place in the sensory and in the secretory epithelium at the time of exposure. The structural changes which follow naturally are not demonstrable for several days.

A comparable sequence of loss of function, followed by structural atrophy and degeneration, is also seen when ultrasound is applied to the central nervous system and has already been referred to.

The same sequence of events was observed by Brain *et al* (1960)—a preliminary engorgement and dilatation of vessels, followed by vacuolation and pyknosis of the sensory cells, and eventually by degeneration of the sensory epithelium and of the nerve fibres, with relatively complete survival of the cupulae and of the tectorial membrane.

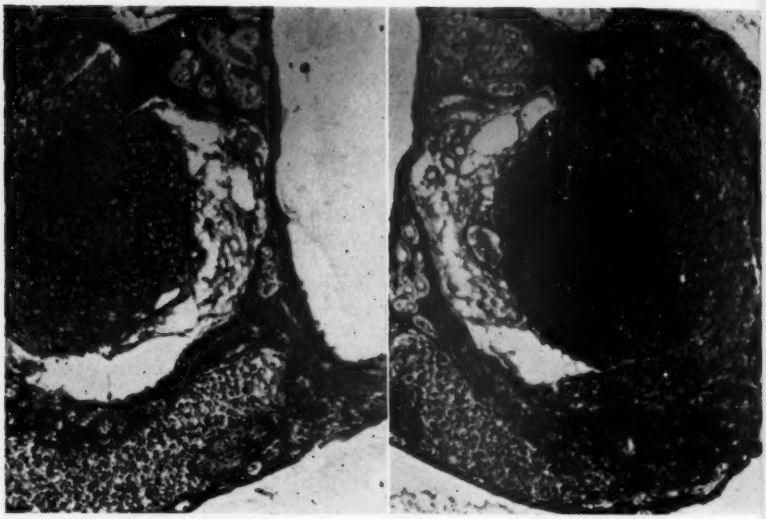


Above: Untreated Right ear × 432.
Below: Treated Left ear × 432.

FIG. 7.

The tectorial membrane is barely altered in shape but the remains of Reissner's membrane lie in contact with it causing an apparent thickening and cellular infiltration of the tectorial membrane, which itself lies in contact with the remains of the organ of Corti. The organ of Corti has collapsed and the tunnel of Corti has disappeared, with the inner rod lying almost on the basilar membrane. The inner and outer hair cells appear to be intact, the latter being displaced bodily towards the basilar membrane. There is not much cellular degeneration, but there is structural displacement due to mechanical forces.

The distance of these changes from the source of ultrasound and the delay in their appearance, suggest that they are due to the mechanical effect of the ultrasound on the individual cells, and not that they are due to the production of heat. If this latter were the case there would be greater disparity between the nearer and the remoter changes and they would appear earlier. The condition of the membranous labyrinth is of interest.



Treated Left ear × 120. Untreated Right ear × 120.

FIG. 8.

There is marked degeneration of many of the cells of the spiral ganglion, and the main trunk of the cochlear nerve shows spaces which represent degenerate nerve fibres.

Shortly after the application of ultrasound there is no change to be seen in the scala media or in the other parts of the endolymph system. At the end of four days there is a dilatation of the utricle, the ampullae and the canals, suggesting that the amount, and the presence of the endolymph has increased. At the end of seven days there is a widespread collapse of the endolymph sac. This is fairly complete in the vertical canal but not in the lateral one nor in the utricle. This suggests that there has been a reduction in the formation of endolymph—or an increased absorption. It does not, in the vestibular area, suggest rupture of the walls of the membranous labyrinth, as if that had occurred there would be total collapse, which is not the case. In the cochlea the membrane of Reissner is ruptured, but if the scala media were emptied of endolymph, rapid increase in the pressure of the perilymph might result in rupture of the membrane, when its elastic power was exceeded. The atrophic appearance of the stria vascularis and the subjacent connective tissue is consistent with a reduction in the amount of endolymph secreted.

It is possible therefore that one biological effect of ultrasound is a preliminary stimulation of endolymph secretion, followed by an atrophic change in the secretory epithelium and a consequently reduced amount of endolymph in the membranous labyrinth.

Histological Changes in the Inner Ear

In the therapeutic application of ultrasound to the human subject it is very important that the following requirements are satisfied.

1. Knowledge of the intensity of ultrasonic force which is emitted from the active face of the applicator, at all times, and which is transmitted to the internal ear.

2. Ability to direct the beam of ultrasonic force to the point at which the maximum degree of effect is desired.

3. Choice of intensity and of duration of application which is sufficient to destroy the function of the vestibular labyrinth, whilst such force, as may be transmitted through the endolymph to the cochlea, remains insufficient to interfere with its function.

Acknowledgements

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THE RADIOGRAPHY OF THE LOWER LATERAL FOOD CHANNELS*

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THE lower lateral food channels extend from the edge of the lateral pharyngo-epiglottic folds downwards and backwards around the laryngeal air passage to the level of the upper border of the cricopharyngeal muscle, where they fuse to become one channel continuous with the lumen of the œsophagus. These channels are functional entities and serve to transmit fluid or semi-fluid substances around the mouth of the larynx, thus preventing spill into the laryngeal airway. During swallowing the bulk of a bolus is usually deflected to either side of the mouth of the larynx and comparatively little passes directly over the edge of the tongue of the epiglottis down the midline: if the bolus is large the larynx is pulled further forwards to accommodate it and two the channels become one posteriorly.

Materials and Methods

During our studies of the mechanism of normal swallowing we have collected about two hundred records of patients with pharyngeal dysphagia. They were referred to us from many sources both in the United Oxford Hospitals and from centres elsewhere. We have also examined a series of about one hundred consecutive patients in the Cowley Road Hospital for old people; not all these patients had dysphagia.

The methods of examination included straight X-rays taken in the lateral position, preliminary fluoroscopic examinations in the usual manner, and cine radiography of swallowing with projections in the lateral and antero-posterior positions and with the head and neck fully turned to the right and to the left.

Our remarks cover the lessons we have learnt from this material.

Normal Findings

In normal young persons during quiet breathing through the nose the lower lateral food channels are either obliterated or reduced to narrow clefts by close approximation of the lower walls of the pharynx to the

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larynx. When films are taken immediately after swallowing barium sulphate suspension there is usually a trace of barium clinging to the mucosa at the sides of the larynx which may outline channels running downwards and backwards to small pouches situated laterally: these are the pyriform fossae. All traces of barium have usually been expressed from the channels below this level. When breathing through the mouth the soft palate and posterior pillars are raised and the lower lateral food channels form air-filled furrows around the entrance of the larynx. If the larynx is partly closed as in speech, these furrows are broadened and extended downwards to the level of the pyriform fossae.

If the pharynx is distended with air as in the Valsalva experiment the pharyngeal cavity is increased in depth and the lateral food channels are opened and partly taken into the lumen of the pharynx. The channels below the level of the pyriform fossae are parted to allow air to enter them to the level of the upper border of the cricopharyngeus: the arytenoids and the body of the cricoid cartilage are moved forwards; permitting the two channels to communicate posteriorly; the pyriform fossae lose their triangular or pyriform shape and become two shallow depressions in the lateral aspects of the floor of the distended pharynx. As the pharynx returns to the state of rest the lower channels below the level of the pyriform fossae are obliterated from below upwards: air or fluid contents are displaced upwards.

Another feature of the distended pharynx is the formation of a small protrusion in the lateral wall—one each side. These protrusions are made in the gap between the middle and inferior constrictor muscles immediately above the lamina of the thyroid cartilage and anterior to the attachment of the stylopharyngeal muscles. They are normally quite small and disappear when the larynx returns to the position of rest.

In old persons the lower lateral food channels are frequently not obliterated at rest as in young people, and may contain saliva or food residues.

When swallowing in the erect position a bolus is usually directed down the midline of the tongue to the level of the median glosso-epiglottic ligament where it is deflected into the vallecula on either side. The epiglottis is tilted backwards towards the posterior pharyngeal wall and serves to arrest the head of the bolus as it descends upon the back of the tongue. As the bolus accumulates upon the epiglottis it spills over the lateral pharyngo-epiglottic folds into the food channels on either side of the mouth of the larynx and passes downwards into the pyriform fossae: at this stage a small quantity of fluid may spill directly over the edge of the epiglottis over the open entrance to the larynx. As the bulk of the bolus enters the upper pharynx the larynx is raised towards the hyoid bone, the contents of the pyriform fossae are expressed upwards and backwards, and the lower part of the lateral channels is opened to

allow the bolus to spill downwards and backwards towards the midline: the columns meet over the back of the cricoid cartilage at the level of the upper border of the cricopharyngeus which then opens to allow the bolus to enter the œsophagus. During the descent of the two streams below the epiglottis some fluid frequently passes behind the arytenoids joining the two columns; this path is used whenever one side is partly or completely obstructed below this level.

When the bulk of the bolus has entered the upper mesopharynx the tongue moves backwards towards the posterior pharyngeal wall and meets the contraction due to the pharyngeal constrictors. The tongue of the epiglottis is gradually displaced with the bolus and is bent downwards at the sides so that it is arched like a monks' hood over the larynx. Each half of the epiglottis serves as a chute to deflect the bulk of the bolus to either side of the midline. A certain amount of the bolus is always displaced down the midline, but this proportion of the total bolus is small unless the amount swallowed is comparatively large, when room is made to accommodate it by taking the larynx further forwards. As the main mass of the bolus is expressed from the mesopharynx the soft palate is lowered and applied to the tongue, and the posterior pillars of the fauces are also brought forwards. The tongue of the epiglottis is pressed downwards over the mouth of the larynx until its tip is over the back of the cricoid cartilage. As the tail of the bolus is expressed from the pharynx the larynx is lowered: the lateral food channels are cleared from above downwards. Finally the cricopharyngeus contracts and expresses the last of the bolus into the œsophagus. Only a small residue normally remains in the pharynx: this is situated over the base of the tongue upon the down-turned epiglottis in what are now the backward-turned valleculae. When the airway is re-established, as the larynx returns to the position of rest, the tongue of the epiglottis springs upwards and the small residue which remains upon the epiglottis is either retained in the valleculae or spilt into the lower lateral food channels and held in the pyriform fossae.

If a person swallows with the head turned fully to one side the bolus is deflected down the lower lateral food channel on the side opposite that to which the head is turned: the whole of an average size bolus may be swallowed down the one lateral channel. Old people with rigidity of the spine or other lesions may not be able to turn the head enough to obstruct totally the side to which the head is turned. Many instruments such as the test meal tube, the gastroscope and the œsophagoscope may be passed entirely down one side: it is for this reason that there is a risk of perforating the floor of the pyriform fossa with a small rigid instrument.

When a normal person swallows lying supine, the bulk of the bolus is still directed to the sides of the larynx but it takes a relatively posterolateral course: the larynx opening which is situated anteriorly is not

necessarily surrounded by the bolus unless the amount swallowed is large. The course of the bolus is likewise influenced by gravity when the patient swallows lying prone or on one side. The effect of gravity is important when we have to consider the problems of management of patients with palsy.

Suckling

When babies are suckling, food is passed backwards and accumulates between the posterior surface of the tongue and the anterior surface of the soft palate above the valleculae: here it is held until sufficient is collected for swallowing to be initiated. As it accumulates, a small quantity may spill over the pharyngo-epiglottic folds around the larynx into the upper part of the lower lateral food channels. When swallowing takes place the laryngeal airway is closed as the bulk of the bolus passes around the larynx: some of the bolus may be spilt directly over the back of the epiglottis as in the adult.

Chewing

When food is chewed, small quantities pass out of the mouth at the sides of the tongue down the upper lateral food channels towards the valleculae: sometimes a little may spill into the lower lateral food channels. The same happens in older subjects, especially those who make mouthing or champing movements, in whom palatoglossal closure is often not effective and who may have difficulty in initiating swallowing. Food may also be spilt from the back of the mouth in those persons who have received injuries to the soft palate or fauces, as for example after tonsillectomy.

Patients with Dysphagia

These may be grouped into four classes.

1. Patients with pharyngeal webs.
2. Patients with neoplasm.
3. Patients with diverticula.
4. Patients with some degree of pharygeal palsy.

1. *Pharyngeal Webs.* We have found pharyngeal webs in patients from childhood to old age. We have never found webs in "normal" subjects under the age of 60, but they may occur in old people. Most of the patients we have examined have had anæmia for many years: some have developed dysphagia after the anæmia has been successfully treated. The majority were female subjects and the youngest was a girl aged 7 years. We have also found webs in patients with no referable symptoms who had dysphagia or dyspepsia due to other causes, but in each case there was usually œsophagitis. Webs have also been found in patients suffering from chronic neurological disorders: it appears that a pharyngeal web is the most common cause of dysphagia in patients in whom dysphagia is not normally

to be expected as a feature of the neurological disease: e.g. we have seen several patients with disseminated sclerosis with dysphagia who proved to have cricopharyngeal webs or neoplasm. All patients with cricopharyngeal webs do not manifest anæmia: it is said that some have a low serum iron level.

Patients with pharyngeal webs usually complain of discomfort in the region of the larynx, typically at the level of the cricoid cartilage but sometimes located to one side. The discomfort or difficulty does not necessarily occur every day, and in some patients there may be a complete remission of symptoms for many years. In some instances the patient may present as an emergency with sudden inability to swallow foods, solids or liquids. One such patient was an old lady of 80 years who gave the story of a previous incident 20 years before.

Radiographic findings: The most frequent site for webs to occur is at the level of the cricopharyngeus muscle, but similar appearances may be seen in one or both of the lower lateral food channels or in the upper œsophagus. Two or more webs at different levels may occur in the same patient. Those which are found at the level of the cricopharyngeus may be seen on the posterior or anterior wall of the lumen, but in some instances are situated more on one side than on the other.

Webs are not always at once apparent and in some patients they may be difficult to record with still radiographs. There are three reasons for this. Firstly they are often not seen unless the channel in the region of the web is moderately distended with barium; this is the chief reason why the relatively inexperienced examiner fails to demonstrate them with still radiographs. Secondly, it is necessary to take films in both antero-posterior and lateral projections since the web can be totally obscured by the mass of the bolus in one of the projections: this is especially important for the detection of webs of the lower ends of the lateral food channels. Thirdly, it is by no means certain that the web is always present: we have seen several patients in whom there has been evidence of a web which we have failed to find at a second examination conducted after a short interval. This observation indicates that webs are due, at least in part, to spasm of muscle and not solely to fibrous stricture.

Webs may be found without any other associated abnormality in the pharynx or upper œsophagus but in some cases there is evidence of small irregularities in the walls of the swallowing passage in the region of the lower end of the lateral food channels and at the level of the cricopharyngeus: sometimes these changes are sufficient to simulate early carcinoma. Some patients show disordered muscular contractions of the lower pharynx.

2. *Neoplasm.* A neoplasm arising in one of the lateral food channels may manifest with dysphagia, a change in the voice, local discomfort

or pain, pain referable to the ear or a hard stony mass in the neck. They are seldom diagnosed in the early stages.

Dysphagia does not occur until the disease is far advanced. This is because a growth arising on one side does not necessarily interfere with swallowing: if one side is blocked the food passing down that side is mostly diverted across the midline to the normal side. Dysphagia is usually not experienced until the growth is large enough to prevent the normal movements of the laryngeal cartilages and hyoid bone in swallowing or until it has extended downwards and medially to invade the opposite side. In exceptional cases the growth forms a mechanical block by producing a polypoidal mass. If there is any restricted movement of the laryngeal structures there may be spill into the larynx as the bolus is swallowed; if there is impaired clearance of the pharynx spill into the lower airway results when the larynx is re-opened.

Change in voice occurs especially in those cases in which the growth is situated on the medial side of the lateral food channels involving the aryepiglottic folds, but there may be no symptoms until the growth is big enough to impair function, either because of its size or by deep infiltration. It is such cases which tend especially to cause local pain or discomfort or pain referable to the ear.

Radiologists may see patients referred with a mass in the neck for examination to exclude calcification in glands and find that it is necessary to keep the possibility of neoplasm of the pharynx in mind, especially in patients in the older age groups: they usually have symptoms referable to the pharynx but these are not necessarily mentioned.

Somewhat similar symptoms to those of carcinoma arising in the lateral food channels may be given by a neoplasm of the upper œsophagus. These cases usually do not have dysphagia in the early stages: in the later stages they may mimic a pharyngeal carcinoma either by becoming fixed and thereby preventing the normal ascent of the pharynx and œsophagus in swallowing, or by spreading upwards to involve one or both of the lower lateral food channels. Post cricoid carcinoma in women is usually situated at the mouth of the œsophagus thereby blocking both lateral food channels and causing symptoms relatively early.

The radiographic changes of neoplasms are deformity, a filling defect, absence of movement, fixation and obstruction. None of these signs is diagnostic of neoplasm in the early stages of the disease but they serve to draw the observer's attention to a particular level in the swallowing way: other projections may be carefully scrutinized for supplementary evidence but it is usually necessary to view the site by direct inspection through an endoscope. In the later stages of the disease the diagnosis is quite obvious by radiographic methods: there are few conditions which simulate carcinoma and none of these is of practical importance.

3. *Pharyngeal Diverticula*. There are several sites where diverticula

may form in the pharyngeal wall, but in only two of these do they occur frequently.

The most common site is in the lower lateral food channels at the level of the deficiency in the muscular wall between the fibres of the middle and inferior constrictor muscles, as previously described. In the first instance these are merely weaknesses in the pharyngeal wall: the protrusion occurs during the expression of the mass of the bolus from the mouth. When the bolus is stripped from the mesopharynx a pocket of barium may be left protruding laterally. The pouch and its residue may entirely disappear when the airway is re-established—the contents of the pouch being displaced into the lower lateral food channel. Rarely these pouches become large and exist at rest as protrusions from the upper end of the lower lateral food channel. They are usually air-filled but sometimes contain fluid or food residues. In exceptional cases the pouch becomes very large on one side and may constitute a severe impediment to swallowing. The existence of these pouches or weaknesses may be confirmed by taking films during the Valsalva manœuvre after barium has been given: the distended air-filled and barium-lined pouch can be clearly seen in antero-posterior projections.

The second common situation is at the junction of the lower lateral food channels with the mouth of the œsophagus. The diverticulum is formed by protrusion between the oblique fibres of the inferior constrictor and the cricopharyngeus muscle. Small diverticula in this situation are very common, especially in the older age groups, and they appear to cause no symptoms. About ten patients per year are seen in the United Oxford Hospitals who have symptoms and diverticula: in not all such patients are the symptoms entirely attributable to the presence of a diverticulum. Typically the patient complains of dysphagia localized to the level of the larynx. If the sac is large enough there will be regurgitation of food into the mouth, especially on bending or with pressure upon the neck. A proportion of patients complain of symptoms referable to the larynx or chest and may develop pneumonia: these symptoms may precede those of dysphagia by months or years.

Cineradiographic studies show that in early cases the diverticulum is not always present. There may be no visible abnormality as the head of the bolus is descending through the open mouth of the œsophagus: a protrusion forms as the main mass of the bolus is being displaced from the upper pharynx and may grow larger as the tail of the bolus is expressed downwards. As the lower lateral food channels are cleared, the protrusion may be reduced in size: sometimes the neck of the protrusion is closed by the contraction of the lower oblique fibres of the inferior constrictor upon the cricoid and a residue remains in a pocket. The bulge may disappear and its contents be displaced into the mouth of the œsophagus as the airway reinflates and returns to the state of rest. In later stages the

diverticulum does not empty completely. The cause of the diverticulum is clearly due to deranged function of the pharyngeal constrictors: the cricopharyngeus closes partly or completely before the whole of the bolus has been displaced from the pharynx and the contraction of the pharyngeal musculature upon the body of the bolus causes the protrusion to appear. In many patients the contractions of the upper pharyngeal musculature are partly impaired so that the stripping of the upper pharynx is, in some degree, defective: thus when the pharynx is reinflated some of the residue may enter the laryngeal airway and descend into the lungs.

4. *Pharyngeal Palsy*. Patients with pharyngeal palsy may or may not complain of symptoms. Typically there is a history of sudden onset of dysphagia followed by slow improvement, and in some instances, total remission of symptoms. Many cases have symptoms attributable to the entry of food in the lower respiratory passages.

Method of examination: The examination must be varied to suit the severity of the patient's complaint. In patients with the severe acute symptoms there is often no indication to make the examination since this will not affect treatment, but in some instances it may clarify the nature and severity of the lesion. Whenever there is any doubt, the patient should be examined in bed with a portable X-ray unit and portable cine camera: a sucker should be available to remove saliva and fluid from the pharynx. In the first instance it is sufficient to obtain lateral projections of the region of the larynx with the patient lying supine. When the apparatus has been set up and the operator is standing at ready, from 2-3 ml. of barium suspension are injected by syringe through a polythene tube inserted into the patient's mouth. The camera is started as soon as the injection is finished and a film of about 6 seconds' duration at 25 f.p.s. is exposed. When the examination has been concluded the patient's pharynx is again sucked clear. The films can be inspected wet. Usually two lateral projections are sufficient, but if necessary the examination may be supplemented: there should be no danger of barium passing into the patient's chest. The examination of the films will show quite clearly where the normal mechanism of swallowing is weakened or paralysed, and whether the larynx can be closed. In severe cases the pharyngeal musculature may be totally paralysed: the distribution of barium sulphate suspension in the pharynx and its passage is entirely dependent upon gravity and the quantity of barium suspension injected. If the patient is lying flat it may pass into the nose or œsophagus but it should not pass into the airway unless too great a volume of fluid has been given.

Cineradiography is also the method of choice for the examination of patients during the stages of recovery and for the examination of those less severely affected: the scope of the examination may be as comprehensive as the clinical condition of the patient warrants.

A few patients who have had pharyngeal palsy or weakness recover more or less completely, but the majority only make a partial recovery and develop alternate means of compensating for their disability: some patients who are entirely free from symptoms are quite unable to clear the pharynx properly.

The lower lateral food channels allow the patient with pharyngeal palsy to swallow small quantities of food and to pass it around the mouth of the larynx without seriously inundating the airway. In most patients the epiglottis does not function properly; it usually is tilted downwards on one side thus permitting the bolus to pass, but may cause partial or complete obstruction of the opposite side: it is not displaced downwards with the bolus as in the normal individual. In most instances the patient acquires the ability to clear most of the upper pharynx with the aid of the tongue, but leaves a residue in the lateral food channels.

Patients who sustain total palsy or weakness of the laryngeal sphincteric mechanism appear not to survive very long: we have not had much experience of such cases. The real danger to most patients is the size of the residue which is retained in the pharynx when he re-establishes his airway. If it is too large it will pass down into the lower respirator tract. The factors governing the size of the residue are first, the quantity and consistency of the food stuff swallowed; patients usually learn to reduce (often subconsciously) the size of the bolus they swallow and to avoid certain sticky, hard or tenacious food stuffs. Secondly, the quantity of residue always depends on the degree to which the patient retains muscle power. If power is weak he may be able to clear the pharynx of fluid but may require to swallow water to move solids: it is necessary for him to use every method to reduce the size of the pharyngeal lumen and it is for this reason that some subjects flex and extend the head or turn it to one side. Thirdly, it is important that there should be no obstruction to the efficiency of the weakened muscular powers. In many patients with pharyngeal palsy the inferior constrictor muscles are not paralysed completely. They may develop relative obstruction either at the level of the cricopharyngeus or in one or both lateral food channels. The obstruction may be complete but in other cases, with the descent of the bolus in the mouth, the lower lateral food channels open to allow the passage of part of the bolus but close partially or completely before the bulk of the bolus has been cleared from the pharyngeal cavity. It is such patients that benefit by stretching or section of the obstructing muscles (Kaplans operation). With the relief thus afforded the patient is able to swallow with the aid of gravity and such muscle powers as he retains.

Since the lower lateral food channels and pyriform fossæ are not reduced in size or obliterated as in the normal individual they serve as receptacles for saliva and food residues: if the pharyngeal residue rises

to the level of the interarytenoid notch the individual must try to reduce this by swallowing again. Many patients so afflicted have to make two or more swallowing motions for each bolus prior to re-establishing an airway.

Other conditions: From time to time, especially in old people, we see abnormalities of the swallowing mechanism associated with dysphagia, whose clinical significance is not yet fully understood. This includes patients in whom the epiglottis does not turn down properly and in whom it may offer obstruction on one side: and patients whose cricopharyngeus partially closes prematurely offering some obstruction to the passage of the bolus.

Discussion

We grow increasingly confident as to the value of radiography as an aid in the diagnosis of patients with difficulty in swallowing. The conventional method of still radiography and fluoroscopy may be used to exclude gross lesions, but they are not reliable for the detection of early organic disease such as carcinoma, or for the analysis of the mechanism of neuromuscular disorders. The limitation of fluoroscopy is mainly the question of the time factor: it is not generally realized that the time available to study the passage of the bolus in any one area of the upper part of the swallowing way is less then 0·04 of a second. Cineradiography at speeds of 25 frames per second or faster provides films which can be studied frame by frame or in slow motion.

Our studies have also enhanced our respect for the statements made by patients. In our view the patient is seldom mistaken about the site of his discomfort: this may not necessarily coincide with the level of his primary organic lesion, but it always corresponds very well with the level of functional derangement produced by his lesion; e.g. one of our patients referred with dysphagia localized at the level of the cricoid cartilage showed no abnormality at clinical examination by the surgeon. Cineradiography demonstrated that he had difficulty in expelling the bolus from the lower pharynx into the œsophagus; the reason for this was not at first evident, for there was no evidence of an organic infiltration or paralysis. We noticed that he had deformity and rigidity at the base of his tongue just below the vallecula: the explanation proved to be a carcinoma of the lower part of the tongue. Re-examination of the films disclosed that the lesion made it impossible for him to lower his larynx properly during the phase of clearance of the bolus from the pharynx: this movement, the lowering of the larynx, is essential for proper clearance of the lower lateral food channels: he therefore had discomfort referred to this situation. Likewise it is not uncommon to find a patient with an obstruction in the lower œsophagus or stomach who gives a history of periodic difficulty in swallowing localized at the level of the pharynx: sometimes this is his presenting symptom. If he is examined on a day

when he has no symptoms referable to the pharynx there may be no abnormality found, but when he has symptoms there may be a considerable degree of neuro-muscular inco-ordination of the inferior constrictor muscles, sometimes simulating pharyngeal paralysis: such cases may result from a reflex disturbance of the normal downward gradient of the swallowing mechanism (when the œsophagus is empty) or from attempts to overcome obstruction (when the œsophagus is full).

Of equal importance is the question of obtaining an accurate history at the clinical examination. Direct enquiries may reveal important symptoms which have not been mentioned. We always enquire what foodstuffs are known to give difficulty: milk drinks and milk chocolate frequently enhance the difficulty of patients who cannot clear the pharynx. This is because milk and mucus in the throat results in the formation of bubbles with tenacious walls which obstruct the pharyngeal airway and cause difficulty when the pharynx has to be cleared to re-establish breathing. Very hot or very cold drinks or an irritable substance such as dry toast, may enhance muscle spasm. We are also interested to obtain a story of a patient wakening from sleep with difficulty in breathing; this difficulty being relieved by coughing or by swallowing a drink of water. In such cases a film taken from the side with the patient lying supine usually shows a pool of fluid or mucus upon the posterior wall of the pharynx. The same explanation holds for the development of noises in the throat while the patient is sleeping: the patient's relatives may comment on this.

We have also been impressed with the truth of some of the teachings of past generations of clinical workers; teachings now sometimes ignored in favour of a radiographic or endoscopic approach to the problem. Hippocrates is said to have stated that "If a man cannot swallow when he turns his head he is sure to die". Now many patients' lives could now be saved if doctors were taught to check this point by asking their patients to drink water with their head in different positions. There have been many clinicians who have emphasized the importance of listening with a stethoscope to a patient swallowing: this appears to be good practice though we do not do it. Many of the older physicians emphasize the value of long-term follow up of women complaining of symptoms in the post-cricoid region: neoplasm may develop after many years of discomfort in this situation even though the patient's anæmia has been adequately controlled: in recent years the importance of the family history in these cases has been emphasized.

Radiography: The radiographic changes which are found in the pharynx in early cases of organic disease are primarily those of impairment of normal function. It is therefore most important that the cine-radiography should be of good quality and that there should be an adequate number of projections in various planes. Special attention has to

be paid to the absence of peristaltic movements, restricted movement of the hyoid bone or laryngeal cartilages, deformities of the walls of the channels, or filling defects. None of these features in the early stages is necessarily diagnostic of the nature of the patient's trouble, but they serve to centre the observer's attention upon particular levels in the swallowing way and other evidence may be sought to ascertain the nature of the lesion. It is also easy to institute an adequate follow-up to see whether the abnormality is improving or growing worse. In the late stages of disease the diagnosis is usually quite obvious by radiographic methods. From what has been said above it follows that radiography is necessarily but one part of the clinical examination of the patient. We believe that in most instances it should be made prior to endoscopy.

We have indicated that accurate diagnosis of the nature of the patient's problem is not always possible by radiographic methods alone, and in inexperienced hands there is the liability for observer error. The present tendency to rely mainly on endoscopy seems open to criticism. Endoscopic methods alone usually do not reveal any but the most severe examples of neuro-muscular disorders: the method cannot enable the observer to appreciate exactly how and why the patient experiences his discomfort. Endoscopy is also frequently not reliable in the diagnosis of early carcinoma. We have seen many patients in whom one or more, in several cases up to four, examinations have been made before a neoplasm has been detected. One explanation for this is that it appears that there are some endoscopists who do not realize that the instrument tends to pass to one side of the larynx, and who do not examine both sides. We have also met with a tendency to give judgement based on a single examination even when the observer has noticed some small abnormality. No-one can question the ability of the endoscopists to recognize gross or late cases of laryngeal carcinoma: the problem is to diagnose the early cases correctly. There is one error commonly made by radiologists, which is easily recognized by the endoscopist: the radiologist cannot detect a cricopharyngeal web unless he can obtain films in several planes with the swallowing passage adequately distended with barium. He cannot do this in patients who will not swallow a large bolus. The endoscopist has no such difficulty for most endoscopy tubes are relatively large and the distension of the passage as the instrument approaches the stricture allows it to be recognized easily.

Finally we would like to re-emphasize the importance of not jumping to conclusions when a lesion is found since this lesion may not be the sole explanation of the patient's difficulty. We have emphasized that pharyngeal webs and diverticula of the lower pharynx are very common in the older age groups, but they not infrequently exist in persons who have organic disease in other parts of the alimentary canal, e.g. a patient with symptoms relating to cricopharyngeus web and long-standing anæmia

who had failed to maintain stability under treatment was found to have developed a pyloric carcinoma: she had no dysphagia, and this was the true explanation of her loss of weight and not the swallowing difficulties. Patients with pharyngeal diverticula who have symptoms referable to the lower respiratory tract should be carefully scrutinized for pharyngeal or laryngeal palsy.

Summary

The anatomy and function of the lower lateral food channels is described. The radiographic changes in the early stages of organic disease are primarily those of impairment of normal function. The abnormal features are not necessarily diagnostic of the nature of the patient's lesion but they serve to direct attention to a particular level in the swallowing passage: other methods of examination may be necessary to ascertain the nature of the changes.

Radiography is a simple method of examination in the old, the very sick and infirm; it is safe. It is also a valuable method for periodic scrutiny of difficult cases not adequately diagnosed by other means. Gross lesions can easily be diagnosed correctly, but for this purpose radiography is probably unnecessary. Attention is drawn to the proper evaluation of the patient's symptoms.

Acknowledgements

We regret space will not permit us to discuss adequately the contributions of previous workers. We gratefully acknowledge the help of those clinicians who have sent us patients, especially Mr. Ronald Macbeth and Mr. G. Livingstone.

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CONDUCTIVE DEAFNESS OF CONGENITAL ORIGIN

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Introduction

A CONDUCTIVE deafness with normal otoscopical findings and with an ætiology which is not always quite clear, originates either from:

(1) Fixation of the ossicular chain or,
(2) Discontinuity of this chain or
(3) Different pathological processes of the eustachian tube.

The fixation of the ossicles may not be only of otosclerotic origin, but sometimes also of a congenital one. These facts we have found from time to time during recent years while operating in the middle ear under magnification. To this belong also the final results of a preceding mitigated infection of the middle-ear cleft or after traumas in this region.

Congenital disturbances or a resulting state after middle-ear infections and injuries of this region, they all can be also the reason for a discontinuity of the ossicular chain.

Ætiology

In the ætiological sense we can sub-divide this sort of deafness of not otosclerotic origin in:

(*a*) Acquired cases.
(*b*) Congenital cases.

Under the acquired cases we can count those, which acquired, in childhood different traumas of the skull or which showed direct injuries of the middle ear. Also operations in this region, performed in childhood may be ætiologically important, because they all may produce an interruption of the chain. Slight and repeated inflammations of the middle ear and of the ossicles and also other mechanical, vibratory or chemical irritations may produce fibrotic adhesions in the middle-ear cleft and be the reason for different fixations of the ossicles.

A disturbed embryological development in the middle ear may be the reason for conductive deafness of congenital origin. The final causes of these disturbances are not always definitely clear and known, but we suppose that healed injuries of a fœtal middle ear, different virus diseases, hormonal and enzymatic imbalances of the mother, drug abuse, eclampsia a.s.o. can lead to these congenital changes in the fœtal middle ear.

From the ætiological point of view, a conductive deafness with an intact tympanic membrane can be subdivided for clearness into the following groups:

(1) Otosclerosis.

(2) Congenital disturbances of the ossicles and the middle ear.

(3) Final results of mitigated or even virulent infections of the middle-ear cleft after birth.

(4) Final results after injuries:

(*a*) A disruption of the chain may be the result of a concussion of the skull.

(*b*) The disruption of the chain may also be the result of a dislocation of fracture parts in the immediate neighbourhood of the ossicles.

(*c*) Direct injuries of the content of the middle ear.

Embryology

In the following we intend to describe in short the most important embryological phases in the development of the region of the middle ear and its content: the middle-ear space and the eustachian tube are developed from the first pharyngeal pouch. The ossicles, muscles and tendons in the middle ear are developed from the first and second branchial arch. In the third embryonic week a quick growing of the first entodermal pouch starts which results in a sideways extension. At the same time the first ectodermal groove deepens from the outside and forms later on the external auditive duct. Between these two extensions remains the mesenchymal tissue in form of the substance of the tympanic membrane.

After 6·5 weeks the differentiation of the malleus, incus and tensor tympani muscle takes place from Meckel's arch.

In the seventh embryonic week the differentiation of the musculus and tendo stapedis takes place, the processus lenticularis incudis approaches the head of the stapes.

After 8 weeks the cartilaginous stapes is pressed against the labyrinth capsule. The musculus stapedis is still a mesenchymal cord. Arteria stapedis still present. Malleus still connected to Meckel's cartilage. The incudostapedial joint is formed. Formation of the manubrium mallei and of the fibrotic part of the tympanic membrane from the mesenchyme.

During the twelfth to fourteenth week the crura and the ligament annulare are formed. Lamina stapedialis is developed out of the mesenchyme of the stapes and the vestibular capsule.

After twenty-eight weeks, ossification of the ossicles and disappearance of the mesenchymal tissue in the middle-ear cleft takes place.

During the thirty-fifth week epithelial lining of the middle ear is formed. In the thirty-sixth week birth and definitive state of the middle-ear cleft.

Congenital Disturbances and Anatomical Variations

An incomplete differentiation of the malleus and incus from the first visceral arch can result in a faulty development or even in a complete absence of these ossicles. A false development of the incus, especially of his processus lenticularis is not a too rare event. In this case we have observed that instead of an ossified substance, a fibrotic cord connected the body of the incus with the head of the stapes.

The myxomatous tissue of the middle ear often persists even after birth. Should a mitigated inflammation of the middle ear occur, this mesenchymal tissue can be changed into a fibrotic one, which results again in a fixation of the chain and in deterioration of the hearing. In other causes again of a more virulent infection of the middle ear, a necrosis of the processus longus in early childhood can happen, the tympanic membrane and the other content of the cleft, after recession of the inflammation, staying fully intact.

These and other anatomical variations are found more and more often during the last years when operating under magnification, and in the following we only want to mention the most important.

Observance of the missing of the musculus and tendo stapedialis has no great disturbing influence on the transfer of acoustic waves. An eventual ossification of this muscle and tendo results in a fixation of the stapes and in a varying state of conductive deafness.

A relatively rare occurrence are anatomical variations of the crura but have no special influence on the hearing. Sometimes fibrotic cords or bony bands were observed between crura and promontorium or to the facial canal and this circumstance may also be a reason of various grades of stapes fixation.

The ligamentum annulare may contain inlets of cartilaginous tissue, which may, later on, ossify. This happens usually between 20-40 years of age and these facts may lead again to an ankylosis of the stapes.

A bony fixation of the incudostapedial joint is very rarely observed. A bony triple-union between the malleus, incus and the stapes is also a very rare event. A fusion of the incudostapedial articulation is more seldom found than an union of the incudomalleolar joint.

Only in a very few cases a total congenital separation in the incudostapedial joint was observed, as well as a complete absence of the incus. Such an absence of this ossicle and a partial under-development and loss of ossification of the processus longus incudis have also been observed by us.

A few times a persistent arteria stapedialis was observed. This persistency can clinically lead to tinnitus and in the case of an operation on

the stapes can cause heavy bleeding. Also persistent branches of the arteria vestibularis could be seen.

Congenital variations on the round window, causing loss of hearing were not described until now.

Through not differentiating the lamina stapedialis from the tissue of the labyrinth capsule, a congenital ankylosis of the stapes plate is stated: in such a case no ligamentum annulare is found, no edges of the plate are to be seen, in difference to otosclerosis no vascularization of the bone is present and the plate shows no tinge of blue.

As already mentioned, persistent mesenchymal and myxomatous tissue in the middle-ear cleft after birth in the form of cords and various folds, is not a rare finding.

Diagnosis

The diagnosis or differential diagnosis of a congenital loss of hearing from a conductive type is not always an easy task.

So in cases of distinct unilateral affections the probability of a congenital variation in the middle ear is bigger than in bilateral cases. On the contrary, bilateral affections we are more inclined to consider as otosclerotic processes.

A conductive loss of hearing, persisting from childhood, is more likely to be of the congenital type. Here we would remind, that malformations of the middle ear sometimes may be parallel to other external congenital changes in the region of the 1 and 2 visceral arch.

Anamnestical findings such as injuries, various operations or repeated inflammations of the middle ear, point more to acquired post-natal disturbances.

A differential diagnosis between congenital and otosclerotic changes in the middle ear is sometimes very difficult to state and this to decide is finally, in difficult cases, left only to the exploratory operation.

The progress of the affection has to be stressed as a most important sign and characteristic: in cases where the conductive deafness shows itself only at a later age, for instance in the second or third decade and a progressive deterioration is observed, we are more inclined to consider these cases as otosclerotic affections.

In cases where the parents observe hearing loss from early childhood, it is more likely to be thought of as a congenital affection. Children under five years are seldom sufficiently co-operative and therefore it is very difficult to come to more precise diagnosis. Often for many years the parents have no knowledge of an unilateral deafness of their child and only at school age they are informed by the teacher of this shortcoming. Only round the tenth year we are able to make a more sure diagnosis. When afterwards the doctor establishes an unilateral deafness, only then the parents start intensified observation. Only too often they are misled in thinking that a fictitious progress of the hearing loss is taking place.

Conductive Deafness of Congenital Origin

The detection of an unilateral deafness by children is so difficult, because automatically they compensate through the other sound side and become very good lipreaders.

Records from the Literature

House divides children under 12 years of age, who suffer from conductive deafness with a normal otoscopical finding, in four different diagnostic groups:

(1) Conductive losses of 15-35 db. in the three speech frequencies, which respond well to the usually used therapy.

(2) Losses of 15-35 db. which do not respond satisfactorily to the used therapy, but in adolescence starts spontaneous regression—this corresponds to a hypertrophy of the tubal lymphoid tissue.

(3) Mild conductive deafness, which increases in adulthood. This is equal to otosclerosis.

(4) Marked conductive deafness in childhood (approximately 45-55 db.) and is constant till adulthood. This is apparently due to congenital footplate fixation.

According to House a differential diagnosis of a congenital fixation of the stapes plate is very probable if:

The conductive loss of approximately 50 db. exists from childhood.
The audiographical curve is flat.
The bone conduction is approximately about 10 db.
No progression.
During the operative exploration no differentiation of the plate from the neighbourhood is found. An eventual mobilization of such a congenitally fixed plate is usually a very hard and difficult task.

Therapy

The therapeutical tasks are according to the patho-anatomical local findings, exclusively determined by operative means. Through micro-orthopædical interventions we try to correct the interrupted chain or to loosen some eventual fixation and ankylosis of the ossicles. In cases of under-developed or lost parts of the chain, we use bone splinters or implantation of pieces of muscle or tendon. Sometimes it may be sufficient to shift the interrupted parts of the chain in such a way that they find a new contact.

In suitable cases we perform a stapediopexy. In ankylotic affections of the stapes mobilization of it or of the footplate is to be advised, in other cases we perform disruption of adhesions and fibrotic cords, or we remove bone grips and bands. In cases where there is no other possibility we have to consider fenestration.

These, from the ætiological point of view very different findings of the middle ear, with an intact drum and with a distinct hearing loss, can lead to very difficult medico-legal situations. In these serious cases an explorative tympanotomy under magnification is advisable.

Experiences of Authors

Performing a microscopic otosurgery during the last years, our experience, regarding the patho-anatomical basis, is growing continually and our knowledge in this respect is expanding.

Brown Kelly (1960) refers to his experience in about 100 stapes-mobilizations in which he found the following non-otosclerotic conditions:

4 cases of very thick mesenchymal lining of the processus longus incudis and stapes, which was very vascularized and velvet like.
6 cases of stout and large adhesions between the ossicles and the wall of the middle-ear cleft.
3 cases of total absence of the incus.

Hough (1958) reports, that between 500 cases of transmeatal tympanotomy he found the following abnormalities:

5 cases of absence of musculus and tendo stapedis and of eminentia pyramidalis.
3 cases with no processus longus incudis.
1 case of bone union of the processus longus with the head of the stapes.
1 case of bone union of all three ossicles.
Several times he observed congenital dehiscences of the horizontal part of the facial canal. Often he found mesenchymal and fibrotic cords on the ossicles and on the round window as well as bone bridges from the crura to the promontorium and to the facial canal.
1 case where only one crus was present and
1 case of congenital fixation of the footplate and with connective tissue instead of the processus longus incudis.
House (1959) reports of:
23 cases of congenital fixation of the footplate of the stapes.

Henner (1960) refers to his findings concerning the following abnormalities performing middle-ear surgery:

1 case of a bony band under the stapedial ligament ankylosing the stapes.
1 case of synostosis maleo-incudialis.
1 case of agenesia of the lenticular process and replacement by a fibrous band. On this band ran a branch of the anterior tympanic artery.
1 case of complete absence of the lenticular process.
1 case of agenesia of the crura with a normal mobile footplate.

1 case of absence of the stapedial annular ligament.
1 case of an absent footplate, an absent annular ligament and agenesia of the crura (with a normal drum and external canal).
1 case where the facial canal ran below the cleft of the oval window.

Our reference material concerns a number of 123 transmeatal tympanotomies performed after Rosen during the period April, 1956 to February, 1960, pre-operatively assuming an otosclerotic process. Within this number we observed:

4 cases with no musculus stapedius.
2 cases of dehiscences on the horizontal part of the facial canal.
9 cases where the processus longus incudis and the stapes were covered very heavily with persistent mesenchymal tissue.
1 case of bony fusion of the incus and stapes with a stapes fixation in the oval window.
1 case of bone triple-union of the incus, malleus and stapes with a stapes fixation in the oval window.
1 case of absence of the incus with a lot of persistent fibromyxomatous tissue in the oval and round window niche.
2 cases of fibrotic and non-ossified processus longus incudis with a stapes fixation in the oval window.
1 case of very strong fibromyxomatous adhesion on the processus longus incudis and on the stapes, with an ankylosis of the stapes in the oval window.
1 case of a traumatic interruption of the chain on the processus longus.

Own Case Reports

Case I.

W.A., boy, aged 11 years. Mother reports bad hearing in both ears since earliest childhood, left side worse. Repeated audiograms show conductive deafness on both sides. Anamnestically no inflammations of the middle ear. Normal otoscopic, nose and throat finding. One year ago adenoidectomy, no improvement of the hearing even after politzerization.

May 27th, 1959, exploration after Rosen, left: fibrosis and adhesions on the stapes and in the niche of the oval window (congenital origin?) fixation of the stapes, processus longus incudis replaced by a thin fibrotic cord.

Disruption of adhesions, mobilization of the stapes, tympanostapediopexy. Whilst in the theatre improvement of hearing of about 20-30 decibels, improvement persisting to this day.

Case II.

S. L., female, aged 39 years. Reports bad hearing since childhood, believes deterioration during the last 20 years, especially left. No inflammations of the middle ear in the past. Otoscopy normal on both sides. Audiogramatically conductive deafness on both sides with affections of the octavus. Otosclerosis on both sides? On December 12th, 1958, mobilization of the stapes, left, and

improvement of hearing. Except a fixed stapes no striking finding in the middle-ear cleft here.

February 25th, 1959: exploration after Rosen, right, processus longus incudis replaced by a fibrotic cord, stapes immobile. Mobilization successful, attempt of tympanostapediopexy. Post-operative audiogram right unchanged.

FIG. 1*a*.

Drawing of the situation after left tympanotomy in Case I. Fibrosis and adhesions on the stapes and in the niche of the oval window, fixation of the stapes, processus longus incudis replaced by a thin fibrotic cord.

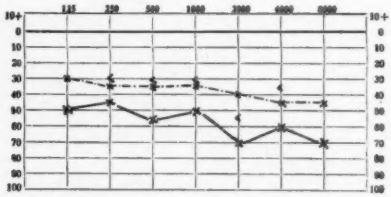

FIG. 1*b*.

Audiogram of Case I: ×———× air pre-operative, ×— . — . —× air post-operative, left < < < bone conduction.

CASE III.

S.S., female, aged 43 years. Bad hearing left since attendance at school, believes deterioration of hearing for 15 years, suffers the last few years also from tinnitus left. No inflammations of the middle ears in the past, otoscopy normal. Audiogram: conductive deafness left. Otosclerosis?

November 20th, 1958: explorative tympanotomy after Rosen left. Bony fusion of the malleus and incus, middle ear filled up with fibrotic and myxomatous tissue. This tissue was partly removed from the niche of the round window and from the stapes, the ankylosed stapes mobilized. Directly after operation improvement of hearing, persevering to the present time.

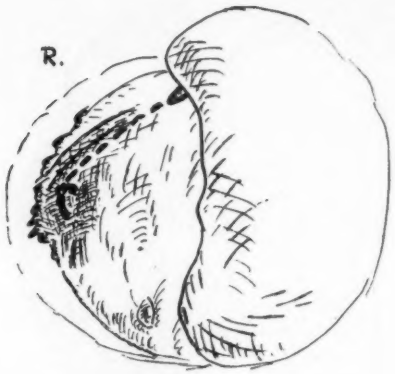

FIG. 2a.

Drawing of the situation after right tympanotomy in Case II. Processus longus incudis replaced by a fibrotic cord, stapes immobile.

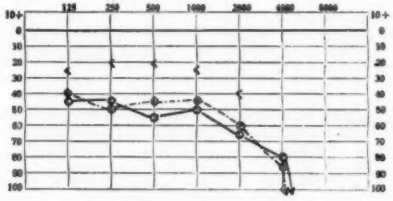

FIG. 2b.

Audiogram of Case II: o———o air pre-operative, o— . — . —o air post-operative, right > > > bone conduction.

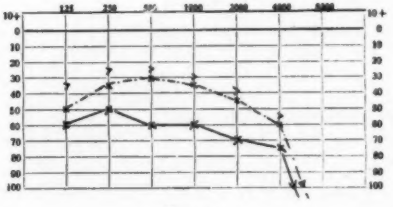

FIG. 2c.

×———× air pre-operative, ×— . — . —× air post-operative, left < < < bone conduction. Audiogram of Case II.

CASE IV.

Z.J., male, aged 51 years. Bad hearing since childhood on the right side. Worked for many years in a weaving shed. For approximately 10 years, observes a more rapid decrease of hearing on both sides. Otoscopical finding

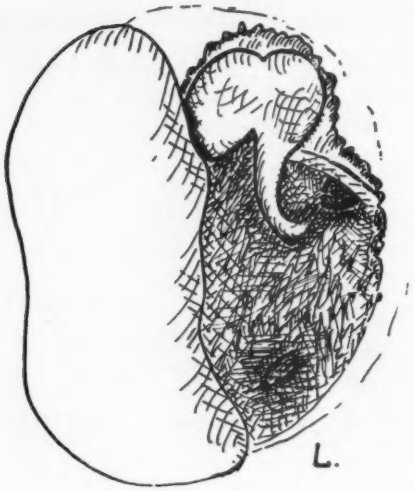

FIG. 3*a*.

Drawing of the situation after left tympanotomy in Case III. Bony fusion of the malleus, and incus, middle ear filled up with fibrotic and myxomatous tissue. Stapes fixed.

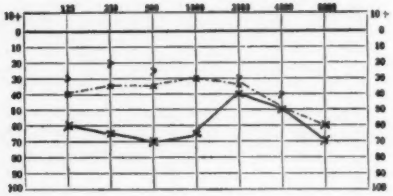

FIG. 3*b*.

Audiogram of Case III: ×———× air pre-operative, ×— . — . —× air post-operative, left < < < bone conduction.

normal on both sides. Audiogram: conductive deafness right with strong sensoneural affection of the octavus on both sides. Otosclerosis right with bilateral nerve affection?

June 17th, 1959: explorative tympanotomy after Rosen right. Bony triple-union of incus, malleus and stapes. Chain and stapes fixed. During attempt to mobilize the stapes, crura and processus longus incudis broken. Post-operatively no change in hearing.

CASE V.

E.E., female, 24 years. Reports bad hearing since school days on both sides, good lip reader, observed deterioration on both sides since puberty. Mother suffering from otosclerosis. Audiogram: very progressive conductive

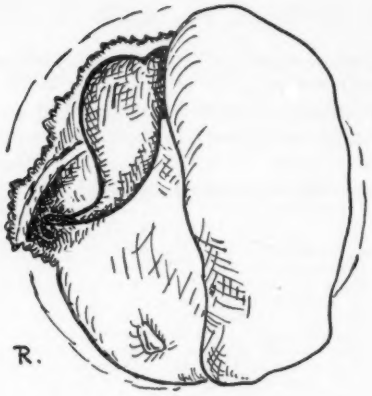

FIG. 4*a*.

Drawing of the situation after right tympanotomy in Case IV. Bony triple-union of incus, malleus and stapes. Ossicle chain and stapes fixed.

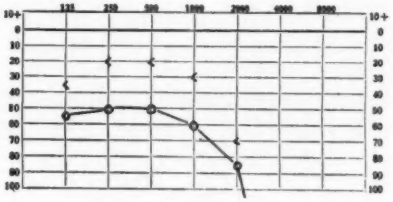

FIG. 4*b*.

Audiogram of Case IV: o———o air pre- and post-operative, unchanged right > > > bone conduction.

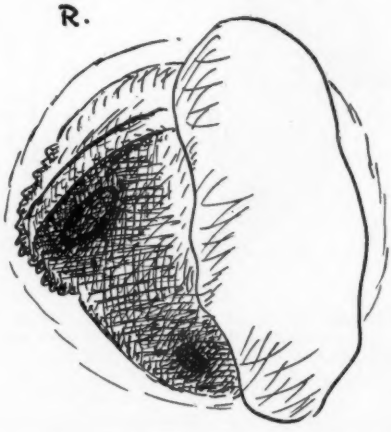

FIG. 5*a*.

Drawings of the situation after tympanotomy in Case 5. Absence of incus in the middle-ear cleft, no stapes established in the compact fibrotic tissue filling up the niche of the oval window. Round window also covered with fibromyxomatous tissue.

deafness with nerve participation on both sides. No inflammation of the middle ear in anamnesis. Otoscopy normal.

December 16th, 1958: right exploration after Rosen. Absence of incus in the middle-ear cleft, no stapes established in the compact fibrotic tissue filling up the niche of the oval window. Round window also covered with fibromyxomatous tissue. Attempt to loosen adhesions failed.

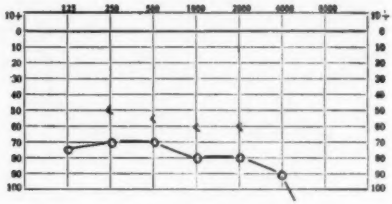

FIG. 5*b*.

Audiogram of Case V: o———o air pre- and post-operative, unchanged, right > > > bone conduction.

CASE VI.

P.C., female, 37 years. Bad hearing since childhood, believes to hear worse for last 10 years. During the last years tinnitus on both sides. Anamnestically

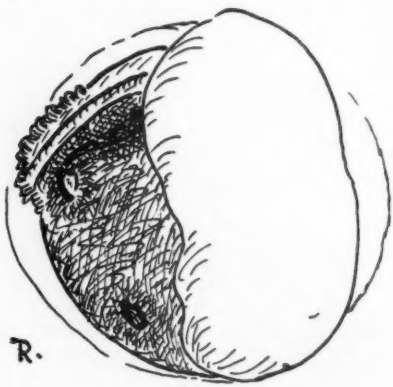

FIG. 6*a*.

Drawing of the situation after tympanotomy in Case VI. Strong adhesions to processus longus incudis and to stapes. Fibrosis and myxomatosis of the niche of the round and oval window. Immobilization of the stapes.

no inflammations of the middle ear. Otoscopy normal on both sides. Audiometry: conductive deafness on both sides with strong affection of the nerves bilaterally. Otosclerosis with affection of nerves bilaterally?

June 10th, 1959: explorative tympanotomy after Rosen right: strong

adhesions to processus longus incudis and to stapes. Fibrosis and myxomatosis of the niche of the round and oval window. Immobilization of the stapes. Disruption of adhesions, mobilization of the stapes. Hearing improved immediately after operation. After one year improved hearing persevering.

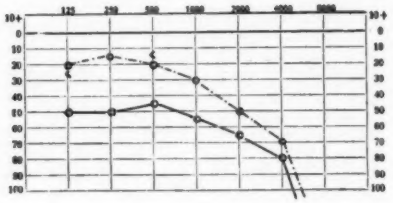

FIG. 6*b*.

Audiogram of Case VI: o———o air pre-operative, o— . — . —o air post-operative, right, > > > bone conduction.

CASE VII.

N.F., female, 24 years. Reports bad hearing on the left side for a long time. No discharge of the ear in the past. Otoscopy normal. Audiometry: a very remarkable conductive deafness left side.

FIG. 7*a*.

Drawing of the situation after tympanotomy left in Case VII. Processus lenticularis incudis partly broken, chain in this place interrupted.

June 23rd, 1959, explorative tympanotomy left: processus lenticularis incudis partly broken, chain in this place interrupted. Attempt to approach the processus longus to the head of the stapes failed. Interposition of a bone

splinter (taken from the rim of the auditory canal) between the stapes and the remainder of the processus longus successful, hearing improved at once.

When asked after the operation, the patient remembers to have been pricked with a knitting needle in the ear by her child some years ago.

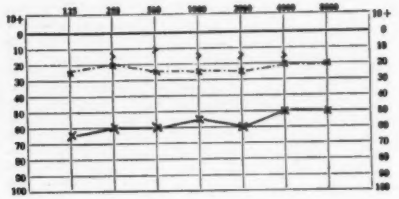

FIG. 7b.

Audiogram of Case VII: ×——× air pre-operative, ×— . — . —× air post-operative, left, < < < bone conduction.

CASE VIII.

C.R., male, 30 years old. For the last 15 years hearing deteriorating increasingly. Never suffered anamnestically from inflammation of the middle ears and never had any head injury.

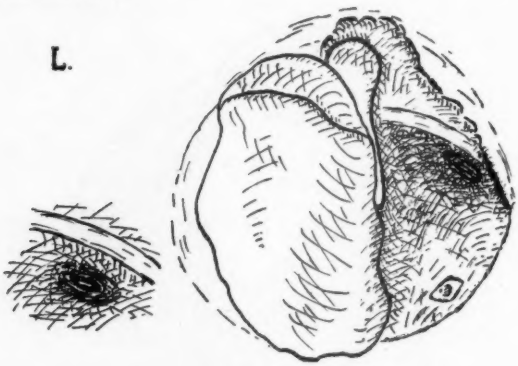

FIG. 8a.

Drawing of the situation after tympanotomy. Absence of incus and stapes with a very small niche of the oval window, with a very small footplate and a bony circular elevation round the footplate. Myxomatous adhesions in the upper part of the middle-ear space. Left: magnification of the oval window niche.

Half a year ago he underwent a stapes mobilization of the right ear because clinically a bilateral stapes fixation had been established. The right-sided deafness of 60 decibels improved at once after operation to 35 decibels. During operation no other abnormality was found but a stapes fixation. On account of this big improvement of the right ear the patient wanted the same operation on his left ear.

On June 20th, 1960, a left-sided tympanotomy after Rosen was performed. Lifting the ear drum we made the following observation: middle-ear space very shallow, ear drum very close to the promontory. The upper two-thirds of the middle-ear space as well as the niche of the oval window completely filled up with myxomatous tissue. No incus and no stapes to be seen. Head of the malleus was present but abnormally formed. After ablation of the myxomatous tissue, the

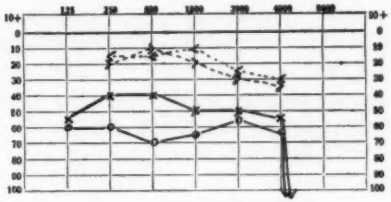

FIG. 8*b*.
Audiogram of both sides of Case VIII: pre-operatively.

niche of the oval window became deep and narrow. On the bottom of the narrow niche a very small foot-plate became visible through the microscope and circular round the foot-plate a bony elevation. After this the ear drum was put back. No improvement of hearing after operation.

This case again shows a congenital absence of the incus and stapes with a conspicuously narrow niche and an abnormally small footplate with a persistence of myxomatous fibrotic tissue in the middle-ear space.

In connection with the otosclerotic process on the right side the bony circular elevation round the small footplate on the left side may perhaps be also explained with a similar otosclerotic process round the small footplate. Therefore in this case an otosclerotic process could have taken place later on in an established congenital abnormality of the middle-ear space.

Conclusion

From our material we have here 7 cases at our disposal, which, with a normal otoscopical finding show a remarkable and constant conductive deafness and where we originally and preoperatively took an otosclerotic process for granted.

During the operation it was established, except in one case (the traumatical one), that we had to deal with congenital disturbances of various parts of the ossicle chain and middle-ear cleft. These disturbances were of three different kinds:

(1) Fusion of two or three ossicles in a partly still differentiated bony lump and fixation of the stapes footplate in the oval window at the same time.

(2) A complete or partial underdevelopment of the incus or its processus lenticularis.

(3) Appearance of fibromyxomatous tissue round the ossicle chain or in the niche of the round and oval window with very strong adhesions.

We report also one case of conductive deafness as a result of an injured middle ear many years ago and where meanwhile the event had been completely forgotten.

Summary

Ætiology, embryology, patho-anatomy, diagnosis with differential diagnosis and therapy of a congenital conductive deafness has been discussed here.

7 of our own cases, mentioned above, were recorded.

1 case of a forgotten injury of the middle ear, resulting in a conductive deafness, has been added.

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4 Lechamej-Hagetaet-str.,
Haifa,
Israel

CORRECTION

"Epistaxis Mortality", Woolf and Jacobs, February, 1961.

Fig. 1. (p. 118). The lower portion of the first three columns should be hatched instead of the upper portion.

Summary (p. 121). 4th line: For "hypertension" read "hypotension".

CARCINOMA OF THE PARANASAL SINUSES

By D. A. OSBORN and P. WINSTON (London)*

In this country, as in North America, sinus carcinoma is generally regarded as a relatively rare disease, accounting for less than 1 per cent. of all carcinomas. The ear, nose and throat surgeon, however, is only too well aware of its existence because of the problems of treatment and the generally poor prognosis. In the published accounts of this disease technical details of treatment figure today no less prominently than in the past —a clear indication of the concern felt by those involved in the management of such cases.

The present paper is concerned not only with treatment but with various other factors which may have a material influence on prognosis. The presentation of this relatively large series of cases provides an opportunity for the recapitulation of the present position of the disease from all points of view.

Material

The cases on which this study is based were drawn from the patients attending the Royal National Throat, Nose and Ear Hospital between the years 1948 and 1959 inclusive. During this period a total of 238,278 new patients attended the associated hospitals. Ninety-four cases of sinus carcinoma were finally selected for inclusion in the investigation. Excluded were all cases of carcinoma originating in the nasal cavity proper, all carcinomas secondary to primary new growths elsewhere and, of course, all tumours of lymphoid tissue and connective tissue. In all 94 cases the tumour arose either in the antrum or in the ethmoid sinuses and it was thought worth while to distinguish the two sites of origin. On this basis there appeared to be 63 antral and 31 ethmoidal tumours.

Age and Sex Incidence

In Fig. 1 the age incidence is seen to rise sharply to a peak in the sixth decade. Males show a slight predominance which is, however, less than 1·5:1.

Symptomatology

Table I summarizes the frequency of the main symptoms. It will be seen that nasal obstruction was the most frequent symptom closely

* From The Department of Pathology and The Professorial Unit of the Institute of Laryngology and Otology, University of London.

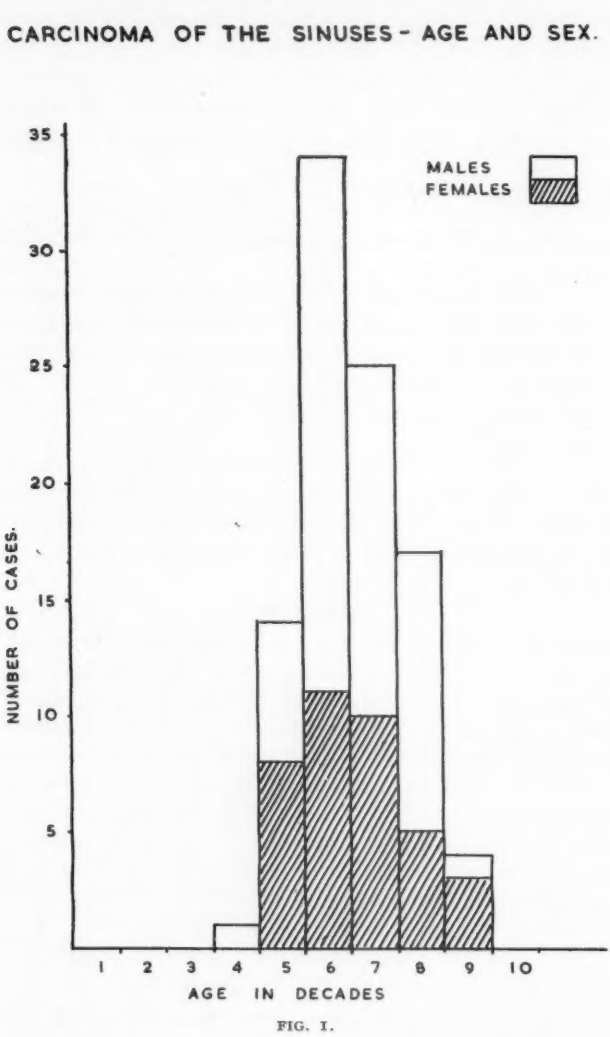

FIG. I.

followed by pain. As might be expected, the former symptom was more closely associated with the ethmoidal tumours, and epistaxis was also more common in this group. In addition, swelling of the face was either noted at examination or complained of by the patient in 42 per cent. of the total, being present in 51 per cent. of antral and 25 per cent. of ethmoidal carcinomas.

TABLE I.

CARCINOMA OF THE SINUSES. FREQUENCY OF SYMPTOMS

Symptom	% Frequency		
	Total	Antrum	Ethmoids
Nasal obstruction ..	53	46	68
Pain	50	49	51
Nasal discharge ..	41	41	42
Epistaxis	38	30	55
Epiphora	22	22	22

It was not possible to determine the precise duration of symptoms in all cases since the inquiry was, to a considerable extent, a retrospective one. However, Fig. 2, which was compiled from such information as was available, gives a fair indication of the scatter in time. The mean duration of symptoms was clearly between one and two months.

Delay in Diagnosis

In the majority of cases the correct diagnosis was made provisionally and quickly confirmed by biopsy, but in 21 cases an initial diagnosis of a non-neoplastic condition was made. In as much as such a procedure might seriously delay the institution of the correct treatment it was obviously pertinent to examine these cases further from the point of view of the delay involved.

In Fig. 3 the time intervals between the date of first attendance and the securing of a positive biopsy are summarized for the 21 cases. Although the delay in establishing the correct diagnosis ranged from 0-13 weeks, it will be seen that in 16 cases the delay did not exceed 3 weeks and in only four cases was it over a month. Taken in conjunction with the duration of symptoms, it is obvious that the delay could have affected the prognosis in only a very few cases.

Treatment

The surgical treatment of these cases was in the hands of a dozen or more surgeons. Radiotherapy, on the other hand, was concentrated in a very few departments of which that at the Royal Marsden Hospital was responsible for the majority. Table II gives a brief summary of the treatment and it can be seen that the majority of cases fall into two main patterns, namely, surgery followed by irradiation and irradiation followed by surgery.

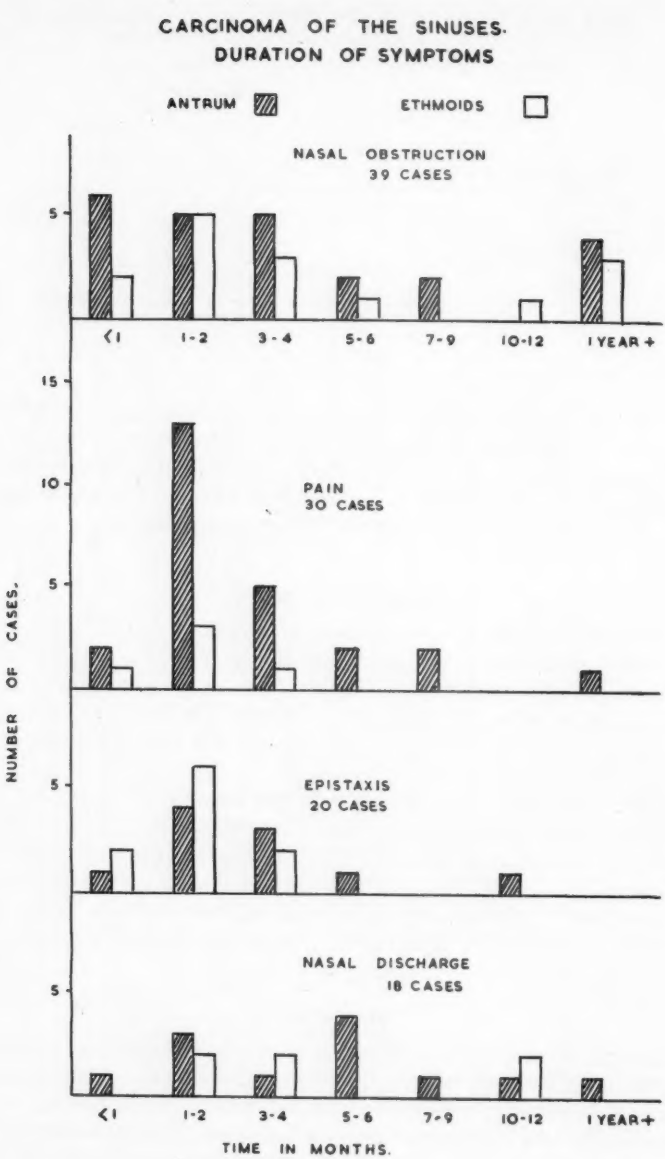
CARCINOMA OF THE SINUSES.
DURATION OF SYMPTOMS
ANTRUM
ETHMOIDS
NASAL OBSTRUCTION
39 CASES
PAIN
30 CASES
EPISTAXIS
20 CASES
NASAL DISCHARGE
18 CASES
NUMBER OF CASES.
<1
1-2
3-4
5-6
7-9
10-12
1 YEAR +
TIME IN MONTHS.

FIG. 2.

INCORRECT INITIAL DIAGNOSIS - DELAY IN ESTABLISHING PRESENCE OF SINUS CARCINOMA.

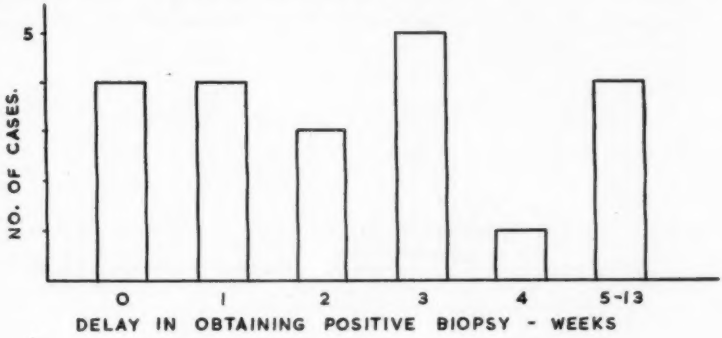

FIG. 3.

TABLE II.

PATTERNS OF TREATMENT.

Surgery followed by irradiation	37	cases
Irradiation followed by surgery	38	,,
Surgery only	2	,,
Irradiation only	9	,,
Palliative irradiation	6	,,
No treatment on account of age and general condition	2	,,
Total	94	

The most frequent surgical procedure was palatal fenestration performed in approximately equal numbers in the two main groups. Excision of the maxilla was carried out in 26 cases, again equally distributed between the two main patterns of treatment. The general pattern of irradiation has already been summarized by Dalley (1957) in respect of those cases treated at the Royal Marsden Hospital.

Results of Treatment

Table III summarizes the survival of cases up to and including 1956. This enables 3 and 5 year survival rates to be calculated on totals of 64 and 48 cases as 33 per cent. and 23 per cent. respectively. The figures for survival without recurrence are slightly less—31 per cent. and 21 per cent. respectively. The crude survival rates appeared to be slightly better for the ethmoidal tumours. It must be pointed out that of those cases surviving for five years or more, one has had a recurrence after 7 years, three have died from recurrences after 6, 7, and 9 years and one had died from an unrelated cause. Analysis of results within the two

TABLE III.

CARCINOMA OF THE SINUSES. OVERALL SURVIVAL RATES.

Period of observation (years)	Total cases in each year	Number surviving	
		Over 3 years	Over 5 years
11	6	3	3
10	3	1	1
9	5	2	2
8	9	3	2
7	9	3	2
6	10	3	0
5	6	2	1
4	8	5	—
3	8	2	—
Percentage survival		33	23

main treatment groups (Table IV) favours slightly the group of cases treated initially by surgery. However, in so far as the surgical measures in this group were frequently non-radical (11 out of 27 cases), it may be argued that any survivals amongst such cases should be credited to irradiation rather than to non-radical surgery. By the same token those cases

TABLE IV.

CARCINOMA OF THE SINUSES. OVERALL SURVIVAL RELATED TO THE TWO MAIN PATTERNS OF TREATMENT

Pattern of treatment	3-year survival		5-year survival	
	Total cases	No. surviving	Total cases	No. surviving
Surgery followed by irradiation	27	13	18	7
Irradiation followed by surgery	22	10	17	4

which received radical surgery after irradiation could be claimed as having been given their effective treatment by the surgeon rather than the radiotherapist. In Table V the cases have been re-grouped into those cases receiving radical surgery before or after irradiation and those cases in which irradiation was preceded or followed by non-radical surgery. The

three-year survival rate now appears to favour slightly the latter group but the five year survivals are identical.

In view of the poor survival rate and the wide scatter in the duration of symptoms it was of interest to examine the correlation between them.

TABLE V.

CARCINOMA OF THE SINUSES. OVERALL SURVIVAL RATES RELATED TO SURGERY AND IRRADIATION

Treatment	3-year survival		5-year survival	
	Total cases	No. surviving	Total cases	No. surviving
Radical surgery before or after irradiation	23	10	14	4
Irradiation before or after non-radical surgery	21	11	21	6

Fig. 4 shows the result of such an analysis on 56 cases seen between 1948 and 1956 for which relevant information was available. The expected correlation is only very roughly shown and a high proportion of the non-survivors had a relatively short duration of symptoms.

Pathology

In the present series gross morbid anatomical studies were restricted by the fact that only twelve cases came to autopsy and only three of these were carried out in this institution. The picture of local spread has, in consequence, been based largely on the findings at operation supported by histological studies on the material removed. In so far as the varied pattern of treatment has resulted necessarily in surgical exploration at different periods, this picture is a complex one in which relative rates of spread cannot be assessed and only an overall impression can be obtained. Table VI summarizes the local spread as so determined. Systemic spread was known to have occurred in 13 cases and 14 cases had clinical evidence of regional lymph node involvement but without histological confirmation in the majority.

Histological classification was based largely on the biopsy material obtained in most cases either by intra-nasal route or by Caldwell-Luc operation. In 15 cases the diagnosis of carcinoma was made on the examination of nasal polypi. Four main histological types were represented as indicated in Table VII. It will be seen that squamous-cell carcinomas predominated and were more common amongst the antral tumours, whereas transitional cell and more especially anaplastic carcinomas had a higher frequency in the ethmoidal group. Adeno-carcinomas arising from sero-mucinous glands showed the structural

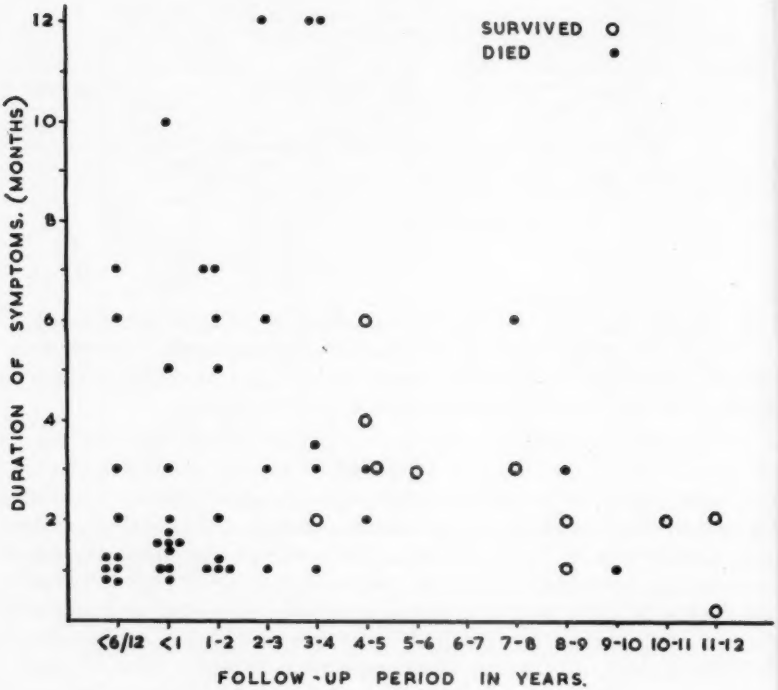

FIG. 4.

characteristics of the "cylindroma" in 3 out of 5 cases. Figs. 5, 6, and 7 illustrate the three main types and of these the squamous and anaplastic carcinomas require no further comment from a morphological point of view.

In this study attention was focussed particularly on the transitional-cell carcinomas which, it will be seen, constituted approximately one-quarter of the total cases. In the present context, these tumours are so-called because of their general resemblance to neoplasms of similar structure arising from the transitional epithelium of the urinary tract. This characteristic they share with the transitional cell papillomas of the nose and sinuses. Their morphology was well described by Ringertz

TABLE VI.

LOCAL SPREAD OF CARCINOMA OF THE SINUSES

Anatomical site	Number of cases		
	Total	Antrum	Ethmoids
Antrum—63 cases	—	—	14
Ethmoids—31 cases	—	18	—
Anterior and lateral walls of antrum and cheek	26	26	0
Antro-nasal wall nasal cavity ..	45	24	21
Palate	16	13	3
Orbit	37	29	8
Frontal sinus	4	1	3
Sphenoid sinus	6	4	2
Anterior cranial fossa	8	2	6
No information	4	3	1

TABLE VII.

CARCINOMA OF THE SINUSES. ANATOMICAL AND HISTOLOGICAL DISTRIBUTION

Histological type	Total		Antrum		Ethmoids	
	No.	%	No.	%	No.	%
Squamous	41	43	35	55	6	19
Transitional	23	24	13	21	10	32
Anaplastic	25	27	12	19	13	42
Adenocarcinoma:						
Simple	2	6	0	5	2	7
Cribriform	3		3		0	
All types	94	—	63	—	31	—

(1938) and more recently by one of us (Osborn, 1956). In the last mentioned paper the characteristic infolding of the epithelium and particularly the persistence of the basement membrane, even in the presence of invasive areas, was emphasized. Two examples of this type of carcinoma were illustrated and a further one is shown in Fig. 7.

FIG. 5.

Carcinoma of ethmoids. Squamous-cell carcinoma in a "nasal polyp". M × 90

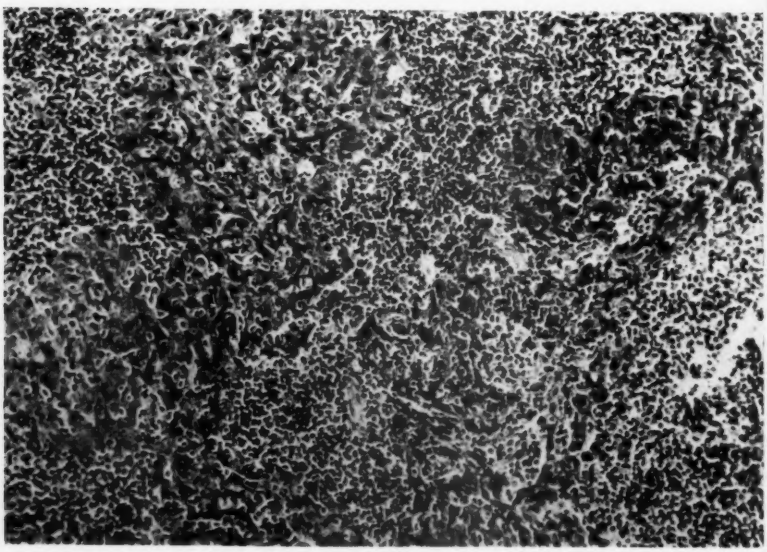

FIG. 6.

Anaplastic carcinoma with marked lymphocytic infiltration—the so-called "lympho-epithelioma" of the antrum. M × 135

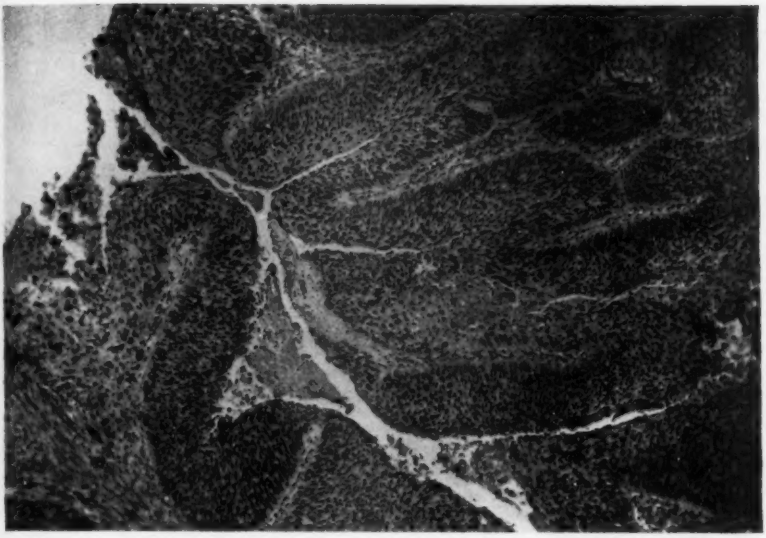

FIG. 7.

Transitional-cell carcinoma of the antrum. The surface proliferation with infolding and persistence of the basement membrane is well shown. M × 56

Fig. 8 shows an example of the type of carcinoma commonly known as "cylindroma". Although of relatively infrequent occurrence, these tumours of sero-mucinous glands are of considerable interest to surgeon and pathologist alike.

Beyond the problems of morphological recognition, interest in histology has centred largely on the possible relationship to prognosis. Table VIII presents the crude survival rates related to histological structure. The dominant position of the transitional-cell carcinomas is self evident but the bare figures are slightly misleading and Fig. 9 gives a truer

TABLE VIII.

CARCINOMA OF THE SINUSES. CRUDE SURVIVAL RATES RELATED TO HISTOLOGY

Histological type	3-year survival			5-year survival		
	Total cases	No. surviving	%	Total cases	No. surviving	%
Transitional cell	17	9	53	11	5	45
Squamous cell	31	9	29	26	3	11·5
Anaplastic	12	2	17	10	2	20
Adenocarcinoma	4	4	—	1	1	—

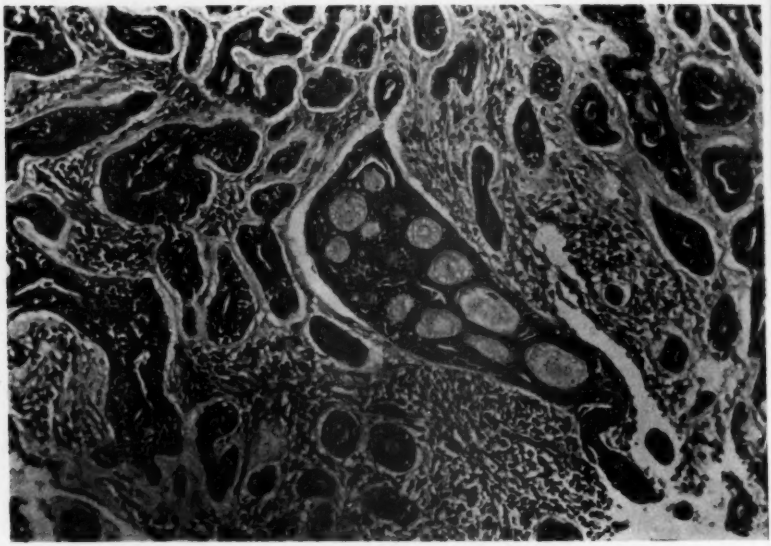

FIG. 8.

Cribriform adenocarcinoma (Cylindroma) of the antrum. The typical central area is surrounded by more easily recognizable glandular structures which leave no doubt regarding its origin. M × 109

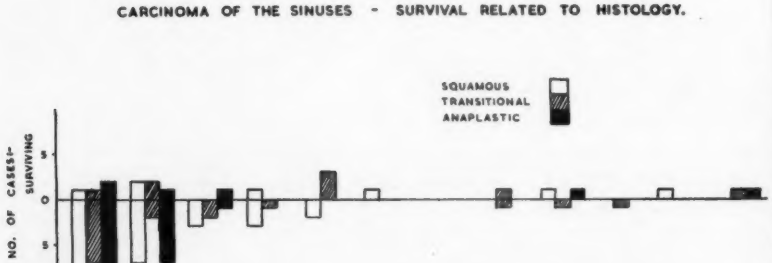

FIG. 9.

perspective. In this diagram, from which the five adenocarcinomas have been excluded for reasons of clarity and simplicity, is recorded the duration of survival at the time of analysis. Above the line are those still surviving and below those already dead. Of possible significance is the fact that out of a total of 9 deaths from unrelated causes in the series as a whole, 5 were in the transitional-cell group. In view of the relatively favourable position of this type of carcinoma, an attempt was made to correlate the fact with the type of treatment. In Table IX survivals in respect of the four histological types are shown in relation to the two forms of treatment as classified in Table V.

TABLE IX.

CARCINOMA OF THE SINUSES. SURVIVAL* OF HISTOLOGICAL TYPES IN RELATION TO SURGERY AND IRRADIATION

Treatment	Trans. cell		Squam. cell		Anaplastic		Adeno-carcinoma	
	3 yr.	5 yr.	3 yr.	5 yr.	3 yr.	5 yr.	3 yr.	5 yr.
Radical surgery before irradiation	5/9	3/4	2/10	0/8	0/1	0/1	3/3	1/1
Irradiation before or after non-radical surgery	3/6	2/6	5/14	2/11	2/5	2/4	1/1	0/0

* Survivals expressed as fractions of the total cases in each group.

Discussion

The present series of nearly one hundred cases of sinus carcinoma is of particular interest because it represents the pooled experience of a dozen or more surgeons often dealing with advanced disease. In a not inconsiderable proportion of cases the duration of symptoms extended into many months. The significance of nasal obstruction is not always easy to assess, but pain, which also figured prominently in the symptomatology, is more clearly defined and, as pointed out by Cranmer (1953), it is, together with facial swelling, an indication of an advanced lesion. Tabb (1957) has commented on the general acceptance of a late clinical picture but its rejection leaves no reliable guide to early diagnosis which remains an unsolved problem.

Erich (1956) and Frazell (1958) have both dealt with errors in initial diagnosis and the consequent delay in the institution of correct treatment. In the present series, whilst delay of this nature might well have been a significant factor in one or two cases, it obviously had no material influence on the outcome in the majority.

With regard to treatment, the present paper has been concerned with the main pattern rather than with details which have been largely

omitted. In recent times, views on treatment have tended to become crystallized into two main schools of thought. Both employ a combination of surgery and irradiation and the chief point at issue is which should be applied first. In the past ten years treatment initially by surgery has been advocated by Schall (1951), Frazell (1955), Struben (1957), Paletta and Weir (1958), Tabb (1959), Hemenway and Lindsay (1959) and Fitz-Hugh and Gorman (1960). First place has been accorded to irradiation by Capps (1950), Larsson and Mårtensson (1954), Snelling and Wilson (1957), Dalley (1957), Castigliano and Rominger (1958) and Hendrick (1958). The exponents of initial surgical treatment often stress the importance of radical excision and Tabb (1957), for instance, considers that radical surgery for carcinoma of the antrum includes excision of the maxilla. In the present series this procedure was carried out in less than one-third of the cases and hence the number of radical operations is relatively small. One might think that by now this issue on treatment would have been settled once and for all by the analysis of results. Unfortunately, it is extremely difficult to assess let alone compare the vast majority of published figures. This is due to a variety of causes:

(*a*) There is lack of conformity over the anatomical site of the tumours reported. Some data are confined to antral neoplasms whilst others include not only all sinus tumours but those originating in the nasal cavity itself.

(*b*) The histology of the tumours is often not taken into consideration. One series may be exclusively carcinomas whilst another includes all malignant tumours of that region. Whilst the vast majority of cases in any series must, of necessity, be carcinomas the distinction is nevertheless important because of the generally accepted better prognosis in non-epithelial malignant tumours of this part of the body.

(*c*) There is considerable variation even within the same pattern of treatment and this is perhaps, the most important single factor in rendering the results obscure.

(*d*) For reasons frequently inherent in the available material, the survival rates are often expressed in terms of varying periods of time which permit no absolute comparison with other published results.

(*e*) A number of survival rates have been published based on selected cases. Whilst the fact of selection is not usually concealed, it is unfortunate that isolated survival rates based on selected "operable" cases tend to be seized upon and quoted against overall survival rates from other sources.

(*f*) Apart from the deliberate exclusion of cases deemed to be beyond treatment, the selection factor may operate in a less controllable

manner influencing the stage at which the patient is presented for treatment. Variation in the stage of the disease is undoubtedly an important factor in the differences between recorded results.

In Table X, which is compiled from the recorded survival rates of the past ten years, an attempt has been made to edit the results in such a way as to render them more comparable. Inability to

TABLE X.

CARCINOMA OF THE SINUSES. PUBLISHED RESULTS OF TREATMENT. 5 YEAR SURVIVALS

Author		Treatment	Site of tumour	Total cases	Survival	
					No.	%
Capps	1950	Primary irradiation followed by surgery.	Nose and sinuses	52	14	26·9
Schall	1951	Varied, but predominantly surgery	Nose and sinuses	202	54	26·4
Mattick and Streuter	1954	Irradiation followed by non-radical surgery	Antrum only	68	7	10
Larsson and Mårtensson	1954	Irradiation followed by surgery ± radical excision	Nose and sinuses	294	68	23
Struben	1957	Mainly surgery followed by irradiation	Sinuses	76	10	13·1
Snelling and Wilson	1957	Primary irradiation followed by non-radical surgery	Antrum and ethmoid	115	32	27·8
Dalley	1957	Various combinations of surgery and irradiation	Antrum and ethmoid	215	61	28·4
Hendrick	1958	Irradiation followed by surgery ± radical excision	Nose and sinuses	68	21*	
Barth *et al.*	1958	Initial surgery preliminary to irradiation	Antrum	34	6	17
Tabb	1959	Radical surgery followed by irradiation	Antrum	60	6	10
Hemenway and Lindsay	1959	Varied. Emphasis on radical surgery in more recent cases	Sinuses	45	6	13

* Survival period 4 to 7 years.

segregate carcinomas from malignant tumours of non-epithelial tissues has led to the rejection of not a few results and even amongst those included many of the variables remain. This applied particularly to patterns of treatment and also to the duration of the disease about which information is not infrequently lacking. Excluded from Table X, for reasons already indicated, are the results recorded by Cranmer (1953), Gibb (1957), Castigliano and Rominger (1958) and Fitz-Hugh and Gorman (1960).

Generally speaking, the tendency in this country is to place the emphasis on irradiation combined with surgery as an adjunct rather than as a radical procedure. In the United States, on the other hand, the emphasis has shifted in recent times towards radical surgery. As yet, this new trend has not produced a sufficient number of cases on which to judge the efficacy of this form of treatment. Many American results cover a period of time when radical surgery was not extensively practised—the time referred to by Fitz-Hugh and Gorman as "the period of palliation and pessimism".

The survival rates in the present series are no better and no worse than many of those previously published, but Table X does require some further comment. Firstly, the results from the Royal Marsden Hospital reported

by Dalley (1957) include a major portion of the cases in the present series. Secondly, the Middlesex Hospital figures (Snelling and Wilson, 1957) are shown here in a consolidated form for the fifteen year period commencing in 1936 when their present regimen of treatment was instituted. Taking their results in five-year periods, the first two had five-year survival rates of 7 per cent. and 8 per cent. respectively whilst in the last period the survival rate was 17 out of 41 cases or 41·5 per cent. This appears to be a considerable improvement in the situation and since there was, apparently no conscious selection of cases, one would like to know whether this is simply due to improved treatment or to other factors such as earlier diagnosis.

The very low survival rates of some of the authors quoted in Table X are undoubtedly due as much to the late stage of the disease as to any other consideration. Thus, in the 68 cases reported by Mattick and Streuter (1954), 75 per cent. were considered to be in a late stage as judged by invasion of the cheek and orbit and secondary deposits in lymph nodes. In the series recorded by Struben (1957) the duration of symptoms ranged from 6 weeks to 4 years with an average of 7 months, again implying a later stage of the disease in many cases. In the present series the average duration of symptoms was certainly less than in the last mentioned author's cases but was still too long. It has been pointed out that Table VI represents the final pattern of spread during the period of observation. An analysis confined to those cases receiving early surgery would enable a truer picture of initial spread to be obtained, but such a procedure did not alter significantly the fact that probably between one-quarter and one-half of the cases already showed involvement of cheek or orbit shortly after coming under observation. This, of course, is in conformity with the symptomatology.

It is obvious that a specialist institution must expect to have referred to it a more advanced type of case, hence, crude survival rates may be correspondingly less satisfactory. Fig. 4 is of interest in showing an almost complete lack of correlation between the duration of symptoms and survival and the implication is clear. As far as the commonly recognized symptoms are concerned, these tumours are clinically silent for a significant period of time. This brings the discussion to the point which has already been stressed by many authors, namely, the importance of early diagnosis. It is well known that in the early stages the symptoms of sinusitis and malignant neoplasia in the paranasal sinuses are indistinguishable and it is not an unnecessary repetition to reiterate the need for early biopsy.

To the pathologist the main point of interest is the possible practical value of histological distinctions between tumours of this region. In this Institution, accustomed as we are to the examination of small fragments of biopsy material, we do not regard attempts to grade these tumours

as being a useful procedure. Moreover, in the face of the dismal prognosis of sinus carcinoma, one may well ask what useful purpose is served by even distinguishing between various types of carcinoma. However, there is evidence, not merely confined to the present investigation, which seems to justify the attempt.

There is a general impression that better results may be expected with adenocarcinomas. Three out of the five tumours of this group showed the histological structure commonly referred to as "cylindroma" or "cystic adenocarcinoma". Recently, Friedmann (1959) has suggested the term "cribriform adenocarcinoma" to describe these tumours of seromucinous gland origin. In the present series, one of these cases survived over five years before finally dying from a recurrence. The other two cases have survived four years, one with and one without recurrence. The experience is typical of this type of carcinoma whose malignant potential is only slightly less than that of other forms. Of the two simple adenocarcinomas, one died within three months and one after 3 years—possibly from an unrelated cause.

Ringertz (1938) described, in great detail, the cylindrical cell or transitional-cell carcinoma and suggested that this group of tumours might have a better prognosis. Relatively little attention appears to have been paid to this suggestion in the subsequent literature, though some authors such as Cranmer (1953) and Struben (1957) have found no useful correlation between histology and prognosis. On the other hand, Larsson and Mårtensson (1954) found a better five-year survival rate amongst the cylindrical-cell carcinomas. The figures could not be described as dramatic, being 36 per cent. as compared with 31 per cent. and 23 per cent. for well-differentiated and poorly differentiated squamous-cell carcinomas respectively. The same authors also found a lower incidence of metastases amongst their cylindrical-cell tumours. Their findings add support to the original suggestion of Ringertz and to the results in the present series.

Perhaps one reason why so little interest has been taken in this type of carcinoma is because, in recent times, it does not appear to have been universally regarded as a histological entity. The term transitional-cell carcinoma was first introduced by Quick and Cutler (1927) and it rapidly became involved in the controversy over "lympho-epithelioma" and it is far from clear what was originally meant by the expression. The subsequent introduction of the term "Schneiderian carcinoma" by Ewing (1929) only served to increase complexity. In more recent times the emphasis has been on simplicity, especially in terminology, and there is no doubt that many transitional-cell carcinomas have been reported as squamous-cell types or, in the case of the less well-differentiated tumours, as anaplastic carcinomas. In an earlier paper (Osborn, 1956) it was pointed out that this tendency to maintain an intact basement suggested a relative reluctance to invade. In other words, there is a

tendency for this type of tumour to remain intra-epithelial, and it would appear that the implication of a better prognosis is supported by the results. In the light of histological considerations it might seem logical to suggest radical surgery as the treatment of choice for this particular type of carcinoma, but it must also be remembered that transitional-cell carcinomas are considered to be relatively radio-sensitive. Although Table IX presents the results slightly biased in favour of radical surgery, the numbers are obviously too small to draw concrete conclusions.

The conclusions which may be drawn from the study of these cases are firstly the need for earlier diagnosis which can only come from increased frequency of histological examination. Secondly, for critical analysis, the results of treatment must be presented in detail with particular reference to the stage of the disease as judged by both symptoms and signs, the anatomical and histological distribution and last but not least the pattern of treatment. It is the last-mentioned which renders the task of comparison most difficult since large uniform treatment groups are not easy to obtain. Thirdly, there is evidence, supported by previous authors, that transitional-cell carcinomas have a better prognosis than the other types, a feature which is apparently inherent in its structure and pathogenesis. This particular group of tumours merits further study particularly in relation to the choice of treatment.

Summary

(1) 94 cases of carcinoma of the paranasal sinuses have been studied.

(2) The crude survival rates were 33 per cent. and 23 per cent. for three and five years respectively. The relatively late stage of the disease in many cases is held partly responsible for the poor results.

(3) Treatment is discussed in terms of the general patterns produced by various combinations of surgery and irradiation. Taking the cases as a whole, no pattern of treatment was outstandingly successful.

(4) Sources of error and difficulty in assessing results of treatment are discussed.

(5) Histology is discussed in relation to prognosis. Evidence is presented to support the suggestion that transitional-cell carcinomas respond better to treatment and so have a better survival rate.

(6) It is suggested that transitional-cell carcinomas should be studied further particularly in relation to the choice of treatment.

Acknowledgments

The authors gratefully acknowledge the co-operation of all the surgeons of the Royal National Throat, Nose and Ear Hospital for access to clinical records. Thanks are also due to Professor F. C. Ormerod, under whose care many of the patients were admitted, and Dr. I. Friedmann,

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A STUDY OF PHARYNGEAL POUCHES

By J. KODICEK and B. CREAMER (London)

FAULTY or absent relaxation of the cricopharyngeal sphincter is, perhaps, the most popular hypothesis to explain the production of pharyngeal pouches. Physiological studies of the pharynx and cricopharynx using the technique of pressure measurements have given an accurate picture of the function of this area in health. We have therefore applied this method to cases of pharyngeal pouch to see if an abnormality of function was demonstrable in these cases.

Methods

The intra-luminal pressures were measured through fine, water-filled polythene tubes attached to Hansen capacitance manometers beside the patient. These tubes were used with their recording holes spaced at 5 cm. intervals. The recordings were made photographically.

Normal Findings

In the two published studies on the normal pharynx and cricopharynx different techniques have been used and somewhat different values for pressures are given. The first study by Fyke and Code (1955) utilized a miniature manometer while the second by Atkinson *et al.* (1957) was with the use of open-tipped tubes similar to our technique.

The pressures in this area are best considered first at rest and then during the act of swallowing. In the resting state the pressure in the pharynx is atmospheric while that in the œsophagus is subatmospheric and reflects the intrathoracic pressure both in level and respiratory swing. In between these areas is a band of high pressure varying from 1 to 4 cm. in length with a mean value of 3 cm. The average maximum pressure in this area is 39 cm. (Atkinson, *et al.*, 1957) or 35 cm. with a range of 10-60 cms. water pressure (Fyke and Code, 1955). This band of high pressure represents the resting tone of the cricopharyngeal sphincter.

On swallowing, a single brief wave of pressure is recorded, travelling in a peristaltic sequence through the pharynx (Fig. 1). The amplitude of this wave ranges from 15 to 80 cm. water pressure (Atkinson, *et al.* 1957) using open tipped recording tubes but an average pressure of 99 cm. of water is given by the miniature manometer method (Fyke and Code, 1955). Synchronous with this wave the pressure in the cricopharyngeal

sphincter drops abruptly from 0·5 to 1·2 secs. This is interpreted as relaxation and opening of the sphincter as the pharynx contracts. The pressure usually falls to intra-œsophageal levels.

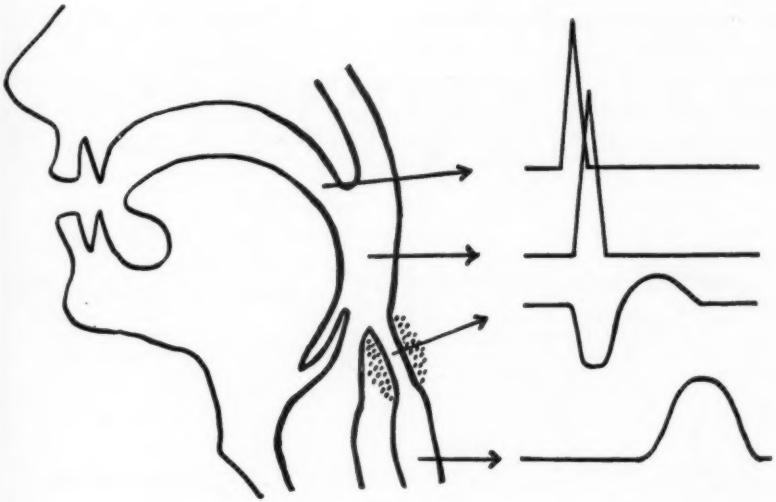

FIG. 1.

A diagram to show the normal pressure changes during swallowing from various sites in the pharynx, cricopharynx and upper œsophagus. The area of the cricopharyngeal sphincter is stippled.

Following this fall, the pressure rises abruptly again and this is frequently followed by a rise in pressure indicating a contraction wave passing through the sphincter and this is propagated down the œsophagus as the peristaltic wave.

The findings in our laboratory agree closely with the reported figures. However, these results have been obtained from healthy young adults and our patients are mostly elderly. Four normal adults of comparable age to the patients were studied to see if age brought about any change in the values. The only difference of note is a rather lower resting pressure in the cricopharyngeal sphincter, range 13 to 25 cm. water pressure.

Patients: Clinical Details

Eight patients with pharyngeal pouches were examined; their ages ranged from 44 to 73 years, the average age being 60 years. Dysphagia was the presenting symptom in all and was of less than two years duration in all but two patients. Regurgitation was present in three cases. The pharyngeal diverticulum was demonstrated radiologically in every patient.

The size varied from a small dimple on the posterior pharyngeal wall in the youngest patient to that of a large pouch.

Results

Passing the recording tubes was extremely difficult in almost all the cases. Radiologically, the tubes could be seen entering the pouch rather than passing through the cricopharyngeal sphincter. Successful intubation of the œsophagus was achieved in five of the eight cases and it is only data from these five cases that is presented here. The results are summarized in Table I.

TABLE I.

	Pharynx		Cricopharyngeal Sphincter			
	Swallowing		Resting		Relaxation	
	Amplitude cm. H_2O	Duration secs.	Pressure cm. H_2O	Length cm.	Amplitude cm. H_2O	Duration secs.
Patient 1	73	0·6	15	3	17	0·4
2	40	0·5	30	3	26	1·0
3	65	0·5	15	—	19	0·5
4	42	0·5	24	3	18	0·5
5	53	0·7	17	4	19	0·5
4 Elderly Patients	40-70	0·5-0·9	13-25	3-4	15-40	0·5-1·0

1. *Resting pressures:* As in health there is a band of high pressure between œsophagus and pharynx. The length in amplitude of this zone is of normal proportions (Fig. 2).

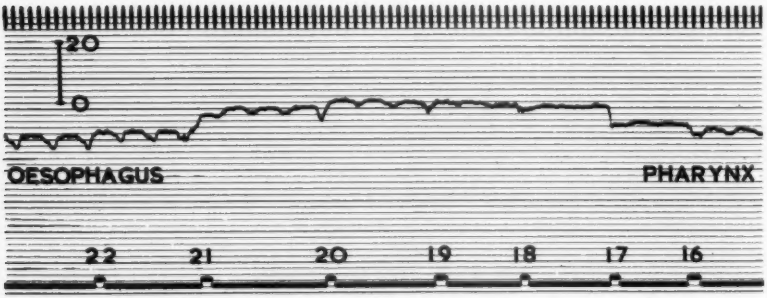

FIG. 2.

Pressure tracing from patient 5 as recording tube is withdrawn intermittently from œsophagus to pharynx without swallowing. At each signal the tube is withdrawn 1 cm. and the distance from the lips marked in cms. The standard is in cms., water pressure and the time marker in seconds. Note the 4 cm. of elevated pressure between œsophagus and pharynx.

2. *Swallowing pressures:* The prime finding is a normal fall in pressure in the high pressure zone on swallowing; the timing and amplitude of the fall are within the normal range except for Patient 1, in whom the duration of the fall was slightly below the normal range. (Figs. 3 and 4) Synchronous with this fall in pressure a wave of high pressure passes

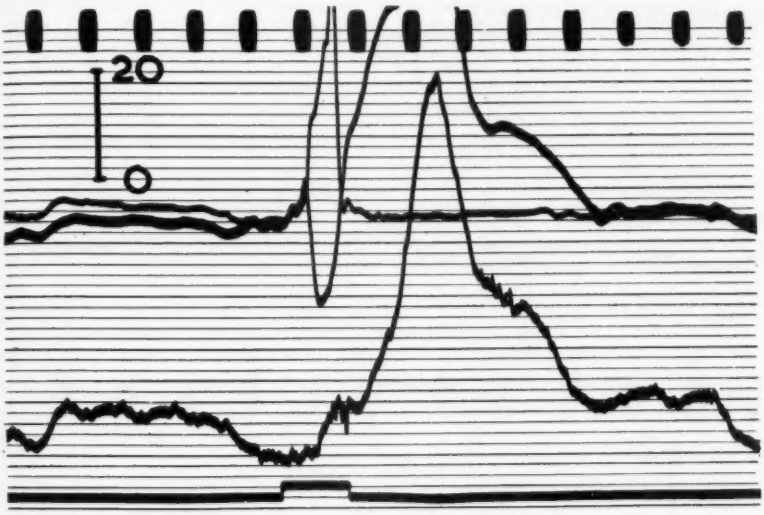

FIG. 3.

Pressure tracing from patient 5 during a swallow. Upper trace from pharynx, middle trace from cricopharyngeal sphincter and lower trace from upper œsophagus. The standard is in cms. water pressure and the time marker in seconds. Note the sharp rise in pressure in the pharynx synchronous with the fall in pressure in the cricopharyngeal sphincter.

peristaltically through the pharynx (Fig. 5). This wave is of normal timing and amplitude.

These pressures indicate that the pharynx and cricopharyngeal sphincter appear to behave in the normal fashion.

Discussion

Pharyngeal propulsion diverticula arise posteriorly between the lowest circular fibres, which form the cricopharyngeal sphincter and the adjacent oblique fibres of the inferior constrictor muscle of the pharynx. A mechanical theory of causation is now widely accepted. It postulates (1) a failure of the cricopharyngeal sphincter to relax in advance of the peristaltic wave of the pharynx and (2) that this failure of the cricopharyngeus to relax anchors the lower pharynx so that contraction of

FIG. 4.

Pressure tracing from patient 2 during a swallow. Position of recording tubes and standards as in Fig. 2.

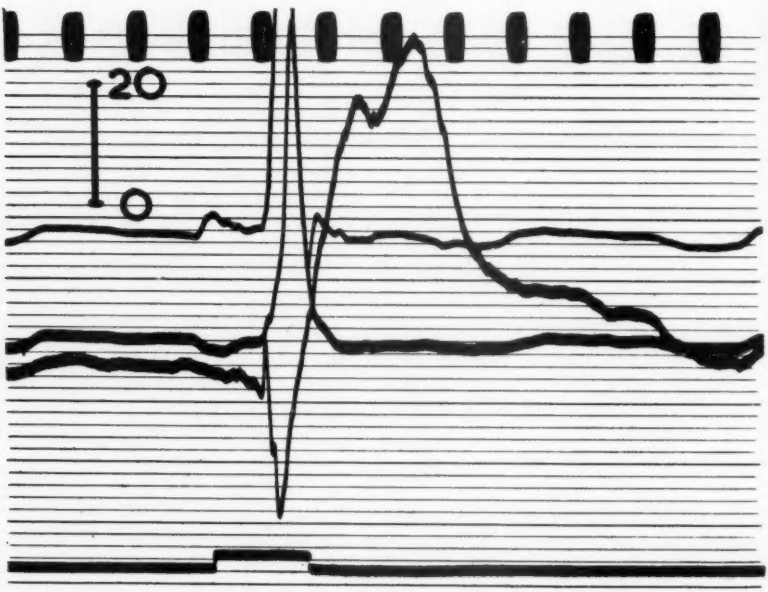

FIG. 5.

Pressure tracing from patient 5 during a swallow. Upper trace from upper pharynx, middle trace from lower pharynx 5 cm. below, and lower trace from cricopharyngeal sphincter. The standard is in cms. water pressure and the time marker in seconds. Note that the pharyngeal wave is peristaltic being later in time in the lower pharynx.

the oblique constrictor fibres pulling in the opposite direction, creates a muscular defect in the posterior wall.

Our observations show that the cricopharyngeal sphincter is functioning normally in these patients and thus make this hypothesis untenable. Pharynx and cricopharynx both show the same patterns as in health so that a disturbance of function as a cause of these diverticula seems unlikely. However, a local inco-ordination of muscle not changing the peristaltic sequence cannot be excluded.

From a clinical point of view there have always been difficulties with the hypothesis, notably the cure of symptoms following removal of the pouch. However, a remission of symptoms is frequently obtained by dilatation of the cricopharyngeal sphincter with a hydrostatic bag and this cannot be explained by our findings.

Summary

The intra-luminal pressures of the pharynx and cricopharyngeal sphincter have been measured in five patients with a pharyngeal pouch. The resting tone of the cricopharyngeal sphinter appears normal and on swallowing normal relaxation is recorded. The action of the pharynx is also normal. No evidence has been found to support a theory of faulty relaxation or achalasia of the cricopharyngeal sphincter as a cause of pharyngeal pouch.

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LARYNGOPYOCELES

By E. GLATZ (Bath)

LARYNGOCELE, the seat of the pyocele, is a pathological enlargement of the appendicular structure of the laryngeal ventricle. This enlargement can be of quite insignificant size and can remain so for a lifetime without causing any symptoms. One finds them inadvertently, though very rarely, in X-rays of the larynx. Once they start to enlarge, they do it in a fashion not unlike a hiatus hernia of the stomach. They can reach considerable size and lead to definite symptoms. These can be purely laryngeal, such as hoarseness, breathlessness, stridor, etc. This happens when the herniation of the ventricular appendix is confined to the intralaryngeal space, i.e. when the enlarged sac lies between the thyroid ala and its intralaryngeal mucosal lining. This variety presents itself as a bulge above the vocal cord. If the enlargement takes the opposite direction, then it burrows along the inner surface of the thyroid ala in an upward direction and eventually herniates through the thyrohyoid membrane and appears as a lateral swelling of the neck. In both varieties the neck of the sac remains short and narrow and terminates in the laryngeal orifice of Morgagni's Ventricle (see Fig. 1).

The usual classification of Laryngoceles is (a) Internal (Intralaryngeal), (b) External (swelling in the neck), (c) Mixed—combined Internal and External. A fully developed Laryngopyocele is of the mixed variety.

Laryngoceles are comparatively rare. Until recently they were rarely encountered, or even thought of by laryngologists and still less by the general surgeon in cases of swelling of the neck. In recent years they have received more attention in the literature and we have become more conscious of their existence. Their symptomatology and treatment have been well defined and their removal does not present any major surgical problem.

Laryngoceles are situated in an area where one would expect to find them exposed to infections following respiratory diseases, or pharyngeal septic episodes. This is not the case. The relative immunity of the sac against infections may in my opinion be due to two factors. One is the presence of the intralaryngeal bulge pressing on the neck of the sac which itself has a narrow lumen, thereby providing a certain degree of protection against the entrance of infected material through the mouth of the laryngeal ventricle. The second factor in a mixed type is that the cervical muscles exert a pressure on the external end of the sac and are

apt to milk back any septic material into the larynx (Fig. 1). This happens sometimes with an audible sound of regurgitation.

Laryngopyocele can presumably develop only, when for some length of time the tissues around the orifice of the ventricle become œdematous

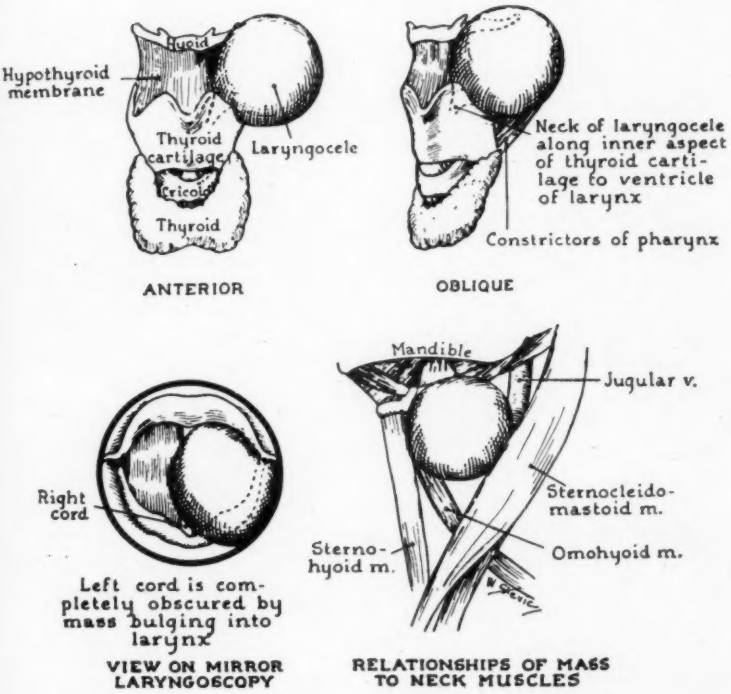

FIG. 1.

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and exert a valvular action against the emptying capacity of the sac. Freedman found in a pyocele granulations at the point of entry from the ventricle and thought that the granulations were damming back secretions in the sac.

Laryngopyocele is said to be an extremely rare disease. Terracol and Guetta (1954) says "While laryngocele is a rare condition, laryngopyocele is a medical curiosity." Once a laryngopyocele develops and is not recognized as such, it can be a fatal disease. The very fact that it is so rare harbours the danger of diagnostic fallacies. To my knowledge there were only eight cases described in the literature, some of them fatal.

Chalier and Dunet (1920), Kistner (1924), Tapia (1932), Zöllner (1932), Terracol (1953), Mays (1954), Chessen and Luter (1955), and Beautyman (1959). As late as in 1959 Beautyman and his co-workers in the summary of their case have this to say: "Occasionally laryngoceles become infected and cause death from asphyxia through emptying of their contents into the larynx, trachea and bronchial tree. The importance of prompt diagnosis and treatment are stressed. It is pointed out that the principal difficulty in the diagnosis of this condition is ignorance of its existence."

One wonders whether laryngopyocele is as rare as that. This seems to be more than doubtful. We have only to look back into the pre-antibiotic past and recollect the cases of parapharyngeal abscesses. They were drained through a cervical incision. Often enough they led to mediastinitis, or septicæmia. I am not at all sure now what was really happening in the parapharyngeal space then, and whether some, in fact, were laryngopyoceles. During the past thirty years the author has had the opportunity to deal with one case of laryngopyocele (see below). It is a good guess that he might have missed some during those three decades. If he did, then he probably was in good company.

Case Report

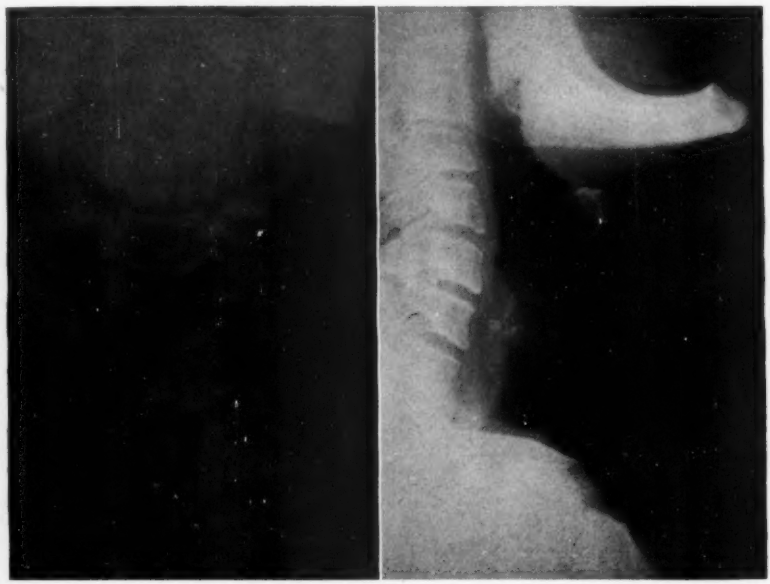
Mr. R. B., 57 years old, was admitted under my care to the E.N.T. Dept. in Bath on November 2nd, 1957. There was a large and painful swelling of the left neck, some stridor and severe dysphagia. There was a previous history of frequent bronchitis. He had noticed a swelling in the left cervical region for about a year. This had enlarged and become tender during the past ten days during an attack of influenza. For the previous 24 hours he was in great discomfort.

On admission: Ill looking man; temperature 101°F.; marked inspiratory stridor; slight trismus and marked dysphagia. Extensive, tender swelling of the left neck. The left hypopharynx bulged with red, smooth swelling. The swelling commenced at the lower pole of the tonsil, filling the left pyriform fossa. The left arytenoid fold and inner surface of the left thyroid cartilage were also bulging, hiding the left vocal cord from view. The entire larynx was displaced to the right. Systemic penicillin and streptomycin were started. Towards the evening the stridor became more pronounced necessitating tracheotomy. Blood Count reported by Dr. H. Gibson, Hb. 105 per cent., Total whites: 10,000, Polymorphs 74 per cent., Eosinophils 2 per cent., Lymphocytes 20 per cent., Monocytes 4 per cent. B.S.R. Westergren fall in one hour = 98 mm. (maximal). Granulocytes showed left shift ++ with toxic degeneration+.

Diagnosis: Parapharyngeal abscess, or secondary infection around a growth. Because of the previous history of a cervical swelling and of

the suspicious intralaryngeal bulge it seemed also possible that this might be a case of an infected laryngocele.

On the following day the intralaryngeal bulge was more marked. The patient was taken to the theatre. In the tonsil position, with a long bladed Davis's gag in place, the intralaryngeal bulge was incised under general anæsthesia using a cuffed and inflated tracheal tube through the tracheostoma. The incision produced a fountain-like gush of pus. The



FIGS. 2 and 3.
X-rays taken two days after evacuation of the Pyocele.

cervical swelling started to collapse and after gentle pressure on the neck emptied itself entirely. Five ounces of pus were produced. A residual flaccid intralaryngeal swelling still remained. The diagnosis of a laryngopyocele seemed almost certain. The X-rays taken two days later clinched the diagnosis. These showed now a large and empty left laryngocele and a rudimentary one on the right side (Figs. 2 and 3). Culture of the evacuated pus grew *Streptococci pneumoniae* ++, resistant to penicillin. The treatment was changed to Tetracycline. The patient made a speedy recovery and was decanulated on the fifth day. Tomographs taken the same day still showed a bulge above the left vocal cord (Fig. 4). He left hospital on the November 20th and was put on the waiting list for removal of his laryngocele.

2.1.58. A bulging on inner surface of left thyroid ala slightly more marked than at the time he left hospital. The left cervical region also appeared fuller than before.

FIG. 4.
Tomograph five days after evacuation.

4.8.58. Excision of a mixed type of laryngocele (Fig. 5). The dissection was easy in the absence of infection.

Report on specimen by Dr. R. Bishton:

Gross: Sausage-shaped thin-walled cyst (6 cm. long × 2 cm. diameter) apparently filled with air. Section taken from fibrous tissue sheath.

Histology: The loose connective tissue external to the sheath shows no malignancy and consists of connective tissue with a few muscle fibres (Fig. 5).

Summary

A case of laryngopyocele is presented. Diagnostic difficulties are discussed. A two-stage operation is suggested; (a) Intralaryngeal incision in the acute stage; (b) Excision of laryngocele in the quiescent stage.

FIG. 5.
Excised Laryngocele.

Acknowledgements

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13 The Circus,
Bath

CLINICAL RECORDS

SURGICAL EXCISION OF A MENINGIOMA FROM THE INFRA-TEMPORAL FOSSA

By C. PETER MILLS (Leeds)

THE following rare case is reported to illustrate an approach to the infra-temporal fossa and the pterygo-maxillary fissure.

A Male Aged 62 Years

Complained of a painless swelling that appeared in the region of his left zygoma two years previously. During the last seven weeks the swelling had recurred, gradually increasing in size and becoming painful. There had been two minor bouts of epistaxis six months before.

On examination—there was a firm, rubbery swelling compressed by the zygomatic arch, occupying the left temporal fossa, and extending into the left cheek, Fig. 1. The mass could be seen and felt at the bucco-alveolar reflection; cross fluctuation was possible between the swelling in the mouth and that in the temporal fossa. The left soft palate had been partially immobilized and distorted by pressure from a smooth swelling involving the left vault and lateral wall of the nasopharynx. The cranial nerves were all intact. There were no other ear, nose and throat abnormalities, and no palpable cervical lymph glands.

Various X-rays of the skull showed the left maxillary antrum to be compressed from without, the left coronoid process and ascending ramus of the mandible to be thinned, Fig. 2.

A soft tissue shadow could be seen extending to the midline of the nasopharynx.

Under a general anæsthetic the extent of the tumour as noted on clinical examination was verified.

An incision was made through the bucco-alveolar sulcus exposing a pale, smooth, rubbery mass that felt to be well encapsulated. It was possible to separate it from the anterior and lateral walls of the maxillary antrum, and the coronoid process. The bone was perfectly smooth. When incising the mass to take a biopsy a small amount of thick whitish mucoid material escaped, this proved to be sterile on culture.

A biopsy showed the tumour to be a meningioma.

Further Investigations

The red and white blood counts were within normal limits. The hæmoglobin was 85 per cent., E.S.R. 17 mm. fall in one hour. W.R. and Kahn tests were negative. The serum proteins were normal.

Operation under General Anaesthesia

The left external carotid artery was ligated above its facial branch.

An incision was made from just in front of the pinna along the zygomatic arch to the outer angle of the orbit, following the lower orbital margin to a point

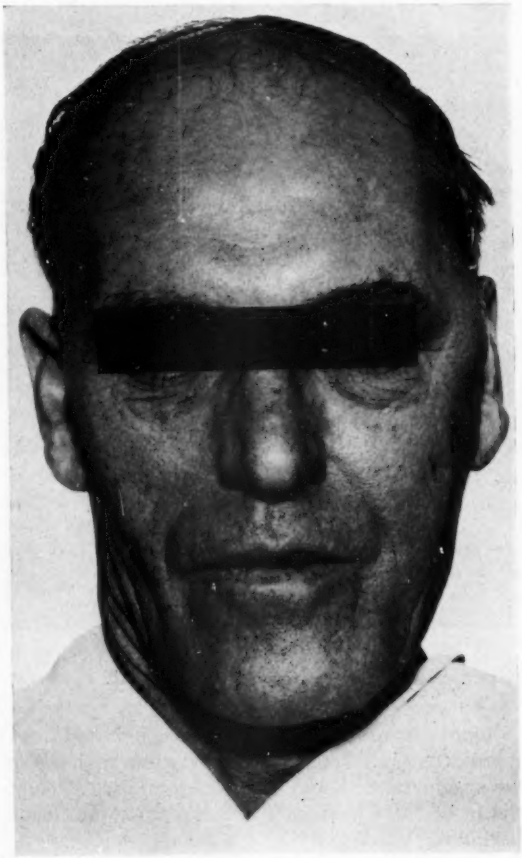

FIG. 1.

Shows a swelling extending into the left temporal fossa compressed by the zygomatic arch.

approximately 2 cm. below the inner canthus, turning along the side of the nose for about 4 cm. The superficial layer of the temporal fascia and the zygomatic arch periosteum were incised.

The periosteum and fascia were then stripped off the arch and the deeper layer of the fascia was divided, exposing the temporalis muscle. A vertical incision through the temporal fascia to the superior temporal line was then

made, which when reflected forwards and backwards, revealed the whole temporalis muscle filling the temporal fossa. The zygomatic arch was divided anteriorly and posteriorly allowing the masseter muscle to be retracted laterally with the bone. The anterior half of the temporalis muscle was detached from the skull anteriorly and retracted posteriorly, exposing the temporal extension of

FIG. 2.

Demonstrates the compression of the maxillary antrum and thinning of the coronoid process.

the tumour. By blunt dissection the tumour was enucleated from the fossa. The inferior and lateral extensions were easily delivered through the space previously occupied by the zygomatic arch and the masseter muscle. The pterygoid plates had been partially destroyed, making removal from the floor of the skull a practicable manœuvre.

A corrugated rubber drain was inserted through the bucco-alveolar sulcus and held with one stitch.

The vertical incision in the temporal fascia was sutured, as was that overlying the zygomatic arch, which served to replace the bone. The skin was closed by interrupted mersutures.

Post-Operative Course

A prophylactic course of terramycin, 250 mg., six-hourly, was instituted for one week.

The drainage was removed after 24 hours and all stitches in six days. The

wound healed by primary intention and apart from a small hæmatoma which was evacuated *viâ* the bucco-alveolar incision, recovery was uneventful.

Two weeks after the operation there was still some trismus. Paralysis of the muscles in the left upper and lower eyelids and the left forehead persisted. Œdema of the lower lid remained for about three months.

Discussion

In 1947 New and Devine read a paper to the American Laryngological Association in which they reviewed 33 cases of neurogenic tumours of the nose and throat encountered in the Mayo Clinic over a period of 27 years. Out of these there were four meningiomas three of which were in the frontal sinuses and one at the root of the nose.

Cushing stated that meningiomas "are rare in the first two decades and reach their maximum in the fifth". When enumerating the sites of origin he said they occurred commonly in the sheath of the trigeminal nerve, and spread widely in the basal meninges, with trigeminal pain and hypæsthesia as early symptoms. They may also invade the orbit from the arachnoid sheath of the optic nerve. He, however, made no mention of them spreading outside the confines of the skull.

According to Brain, meningiomas are very rare below the tentorium. "Commonly the meningioma is a single large, more or less irregularly lobulated growth, but less frequently it may form a flat plaque spreading over the inner surface of the dura."

They are slow growing expansile tumours that usually grow by pushing aside surrounding tissues. Meningiomas do not metastasize but recur locally if not completely removed.

"They are composed of specialized connective tissue cells resembling the arachnoid villus. These cells are present in columns or whorls, and the tumour sometimes contains fibroglia together with collagen fibres and small calcified concretions known as psammona bodies."

Fig. 3 shows the whorling of the endothelial cells in some areas, and interweaving of fasciculated spindle cells in others. Mitosis is inconspicuous, and there is nothing to indicate overt malignancy.

Although a meningioma is an extremely rare tumour at this site, it has some very interesting features.

It seems from the history that it had been present for at least two years—probably much longer, judging from its rate of growth. There had been no symptoms apart from a little pain in the last seven weeks, the main complaint being of a swelling in the temporal region. Speech and swallowing had in no way been interfered with. The X-rays show how it had compressed the maxillary antrum and greatly thinned the ascending ramus of the mandible.

During the preliminary exploration it was seen to be well encapsulated and obviously removable if an approach could be found to give access to its deeper parts.

The incision was designed to give a wide exposure and at the same time preserve as much facial nerve intact as possible. At operation the exposure was adequate, and the cosmetic result six months later was excellent (Fig. 4), the

patient experiencing very little disability from his paralysed eyelid and forehead muscles.

The preliminary procedure of ligating the external carotid artery above its

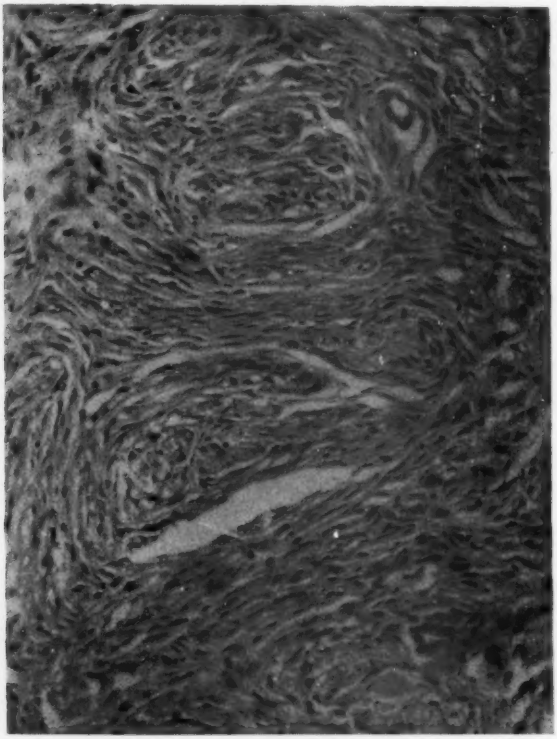

FIG. 3.
Illustrates the whorled effect of the endothelial cells.

facial branch, made the operation relatively avascular. By keeping close to the capsule no branches of the Vth cranial nerve were encountered.

Summary

A case of meningioma in the infratemporal fossa is reported.
An approach designed for its excision is described.

Acknowledgments

I wish to thank Mr. Philip Reading for his kind permission to operate on and report this case. I am also indebted to the photographic department, Guy's

Hospital for Figs. 1, 2 and 4. My thanks are also due to Dr. Denis Harriman of the Pathology Department, Leeds General Infirmary, for the histological report and the photomicrograph.

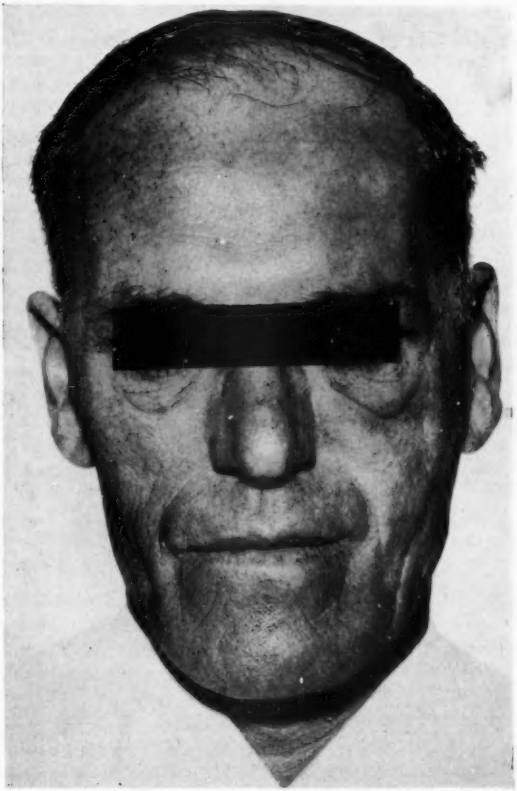

FIG. 4.

Cosmetic result six months after operation.

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DECOMPRESSION OF THE FACIAL NERVE

With an Illustration of Exposing its Vertical Part in Bell's Palsy

By DR. ABDEL SALAM EL BARBARY and DR. HASHEM A. FOUAD
(Cairo)

The idea of decompressing the facial nerve is to get access to the site of damage to the nerve either secondary to trauma or infection or to relieve the intracanalicular pressure upon the nerve in the canal and especially its vessels.

In cases of facial paralysis of otitic origin it is essential to decompress the nerve from the stylomastoid foramen to the geniculate ganglion.

Decompression of the vertical portion of the nerve from the lateral semicircular canal to the stylomastoid foramen is recommended in some cases of Bell's palsy and hemifacial spasm.

Alt was the first to advocate decompression in 1908 when he decompressed the facial nerve above and below an erosion in the canal produced by cholesteatoma.

Nay in 1933 suggested repair within the temporal bone.

In 1930 Bunnell used the aural nerve to bridge a gap of the facial nerve in its canal.

Otology owes a great debt to Ballance and Duel in establishing a definite pattern for surgical intervention in facial nerve paralysis in 1932.

Some modification of their technique was described by others like Sullivan and Kettel.

They exposed the mastoid by a post-auricular incision and a complete mastoidectomy was performed.

The tip of the mastoid is freed from any fibres of the sternomastoid muscle and the posterior end of the lateral semi-circular canal, the proximal landmark is exposed. The tip of the mastoid after being converted to a thin shell of bone is removed, more bone is now curetted till the funnel-shaped stylomastoid foramen comes into view. Now the distal landmark for the vertical segment of the nerve is exposed.

Between these two landmarks the posterior bony wall of the external auditory meatus is shaved down by burrs and curettes. When only a very thin layer of bone covers the nerve, the canal is opened from below upwards using very fine curettes and the vertical segment of the nerve is exposed in its full width. Decompression is done starting from the stylomastoid foramen and working upwards to the lateral semi-circular canal.

Miles L. Lewis described another exposure. He uses an endaural incision, removes the mastoid cortex over the aditus, antrum and mastoid process extending anteriorly until the antrum and short process of the incus are exposed and inferiorly and medially to the digastric ridge. Having thinned the posterior and superior osseus external canal, the tympanic cavity is entered with a burr

1 to 1½ mm. inferior to the lowest extension of the short process of the incus just superficial to the horizontal semi-circular canal and the opening is enlarged circumferentially.

His aim was to expose the Fallopian canal through this opening in its course between the horizontal semi-circular canal and the stapes. He takes this part of the Fallopian canal as a guide and follows it, removing the dense bone overlying the canal with cutting burrs sweeping from above downwards.

Recently Meurman exposes the nerve by a tympanic approach using an incision similar to that used in stapes mobilization reflecting the posterior half of the drum forwards exposing the ossicles and the nerve is followed from above the oval window—down to the stylomastoid foramen.

In cases of mastoid infection we explore the mastoid preferably by an endaural approach yet in cases of Bell's palsy we agree with Meurman that it is not necessary to open the mastoid air cells but we find no reason for opening the middle-ear cavity.

Our technique in exploring the facial nerve in cases of Bell's palsy is as follows:

We perform an endaural incision and not the incision used for mobilization as Meurman describes. We carry the incision in the meatus deeper than the junction of the cartilaginous and bony parts of the meatus so that the reflected skin covering the deep part of meatus will form a small flap giving an easier exposure (Fig. 1). The skin over the outer part is reflected laterally and a three-bladed retractor placed. This exposes the supra-meatal spine and the bony meatus (Fig. 2).

The skin over the deep part of the meatus is reflected medially till the edge of drum, without opening the middle ear.

The mastoid antrum is opened with the burr and the short process of the incus exposed with the rather strong ligament fixing it to the bridge. Widening the antral cavity with burrs exposes the curve between the horizontal and vertical segments of the facial canal. This lies directly below the short process of the incus and the semi-circular canal (Fig. 3).

The facial ridge is lowered gradually and the canal followed downwards to the stylomastoid foramen. This will expose the facial canal in its vertical segment, the area we need explore in cases of ischaemic facial palsy (Fig. 4).

After the pathological lesions in the nerve sheath and Fallopian canal are dealt with, the skin flaps are replaced and the meatus is packed.

Comment

In cases of facial paralysis of otitic origin it is essential to explore the mastoid antrum and mastoid air cells.

In cases of ischaemic facial palsy or cases of hemifacial spasm with no infection of the mastoid cells decompression of the nerve only is advisable.

This can be performed without opening the middle ear as we described.

Summary

A variation in the technique of decompression of the facial nerve in its vertical portion has been described. It does not affect the hearing as the middle-ear structures are not touched, will minimize hospitalization and morbidity.

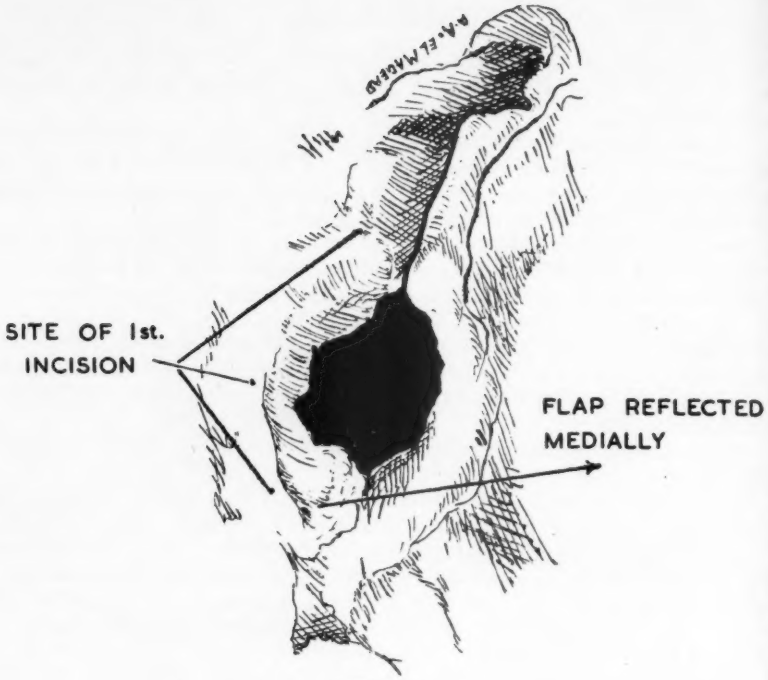
A.A.EL MAGEAD
SITE OF 1st.
INCISION
FLAP REFLECTED
MEDIALLY

FIG. 1.

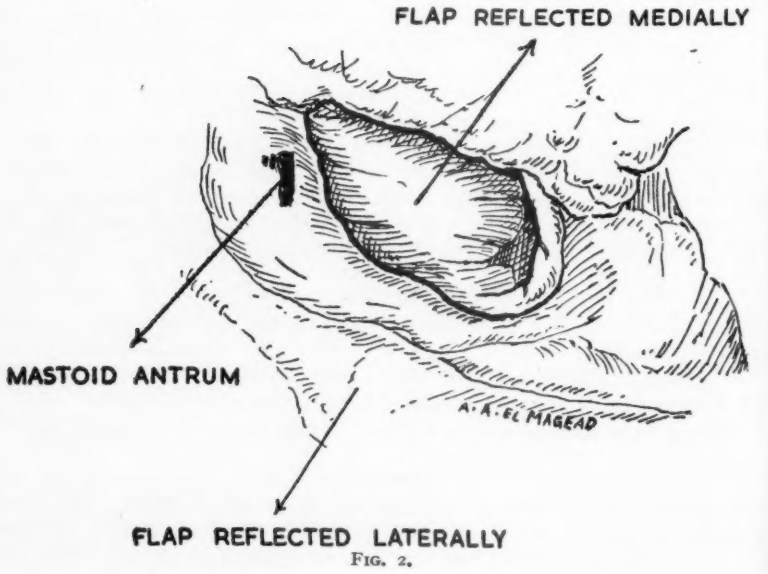
FLAP REFLECTED MEDIALLY
MASTOID ANTRUM
A.A.EL MAGEAD
FLAP REFLECTED LATERALLY

FIG. 2.

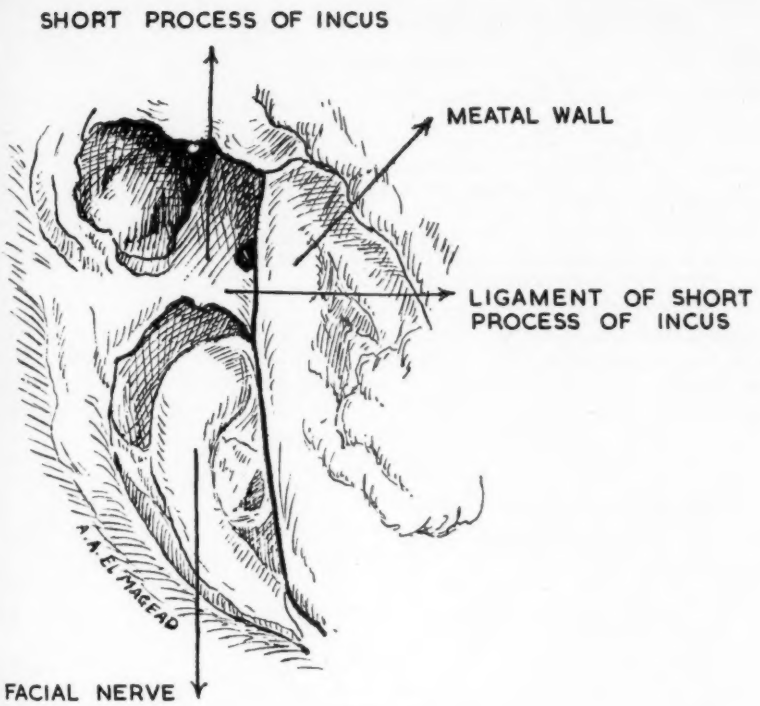
SHORT PROCESS OF INCUS
MEATAL WALL
LIGAMENT OF SHORT PROCESS OF INCUS
A. A. EL MAGEAD
FACIAL NERVE

FIG. 3.

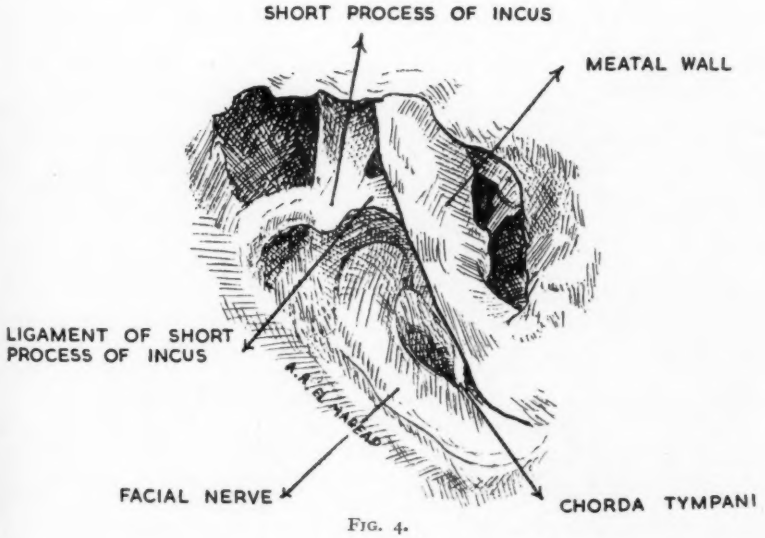
SHORT PROCESS OF INCUS
MEATAL WALL
LIGAMENT OF SHORT PROCESS OF INCUS
A. A. EL MAGEAD
FACIAL NERVE
CHORDA TYMPANI

FIG. 4.

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A CASE OF PRIMARY CHORDOMA OF THE SPHENOIDAL SINUS

By D. F. N. HARRISON (London)

CRANIO-CERVICAL chordoma is uncommon enough to warrant the recording of even a single case. Ormerod (1960) found only one case in the combined records of the Royal National Throat, Nose and Ear Hospital and the Royal Marsden Hospital extending over a period of eleven years.

Case Report

William D., now aged 52 years, was first seen at the National Hospital for Nervous Diseases in *April 1958*. He was complaining of left frontal pain and drooping of the left upper eyelid for the past six days. Examination showed slight ptosis with impairment of upward movement of the left eye. The pupils were normal in size and in their reaction to light and accommodation.

Dr. Hallpike saw the patient and after testing thought that there was some directional preponderance to the left of the optokinetic response. X-ray showed considerable destruction of the posterior clinoid processes and dorsum sellae, the floor of the pituitary fossa showed lack of definition. These radiological findings were thought to be indicative of increased intracranial pressure.

A left carotid arteriogram and a vertebral arteriogram were reported as being normal.

No definite diagnosis was arrived at and the patient was seen on several occasions until *June 1959*. Frequent X-rays were taken but showed little change.

In *April 1960* the patient was seen at the Medical Out-Patients, Guy's Hospital by Dr. C. Joiner. He complained that the headache, present intermittently for the past two years, had become much more severe during the previous six weeks. Examination showed weakness of the left lateral and superior rectus muscles. The left pupil was larger than the right and it reacted more slowly to both light and accommodation.

X-ray showed erosion of the anterior wall and floor of the sella, almost complete destruction of the sphenoidal sinuses, destruction of the adjacent left ethmoidal cells and possibly of the medial wall of the left superior orbital fissure (Figs. 1, 2 and 3).

Carotid arteriograms and an air encephalogram suggested that the structures in and around the sella were being pressed upon from below.

Examination in the Ear, Nose and Throat Department revealed no other abnormality . . . the nasopharynx being quite normal. A tentative diagnosis of a mucocœle of the sphenoidal sinus was made.

On *May 25th, 1960*, the left sphenoidal sinus was approached by means

FIG. 1.

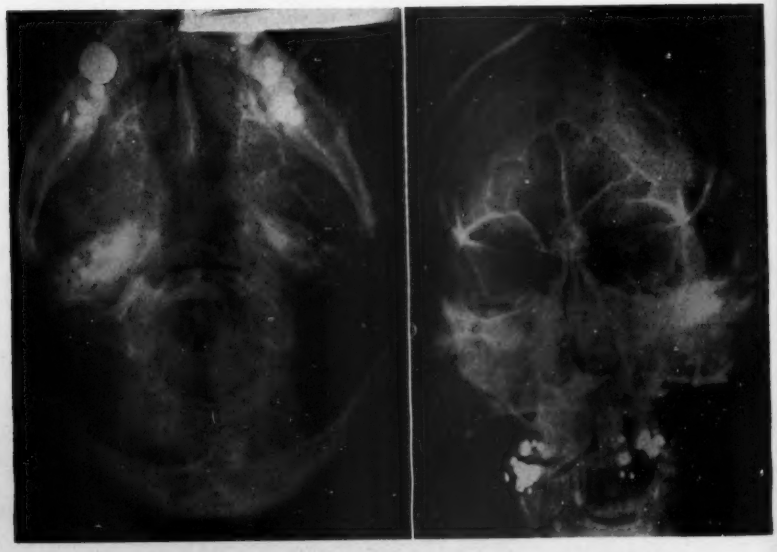

FIG. 2. FIG. 3.

Showing bony destruction by tumour in the Sphenoid.

of a Paterson's transorbital ethmoidectomy. The whole of the anterior wall of both sinuses was found to be replaced by a firm blue cystic swelling. This was ruptured and the mucoid contents aspirated, the cyst wall was removed as far as possible in view of the extensive loss of the surrounding bony walls. When this had been carried out the pituitary gland was found to be hanging free from the region of the sinus roof. The operation was then completed as for a routine ethmoidectomy and the postoperative recovery was uneventful.

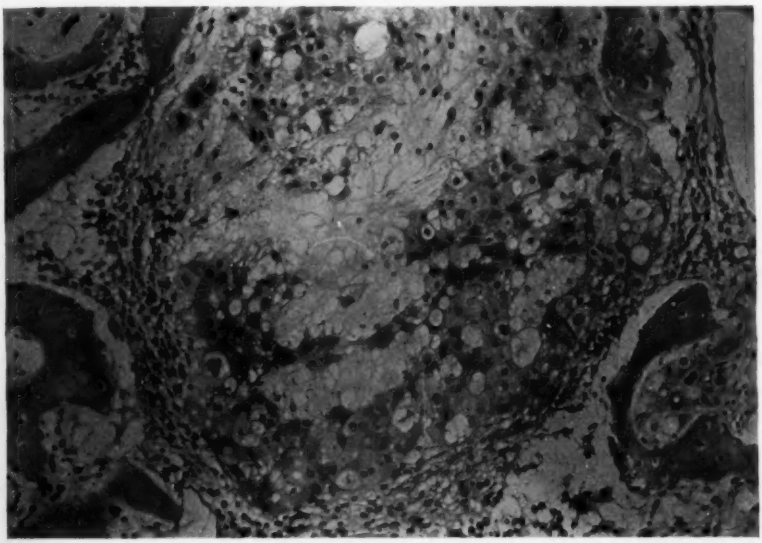

FIG. 4.
Section of chordoma showing vacuolated cells and gelatinous intercellular substance.

Immediately on recovering from the anæsthetic the patient commented on the loss of his frontal pain and within two days his diplopia had vanished with a return to normal of all ocular movements.

The patient has been seen regularly since the operation and there has been no return of symptoms. No claim is made that a radical clearance was carried out, firstly because it was not technically feasible and secondly because the provisional diagnosis of a mucocœle appeared to be confirmed at operation.

The biopsy taken from the cyst wall showed the typical features of a chordoma (Fig. 4).

The clinical and pathological features of cranio-cervical chordoma have been well described by Stewart and Morin (1926) and more recently by Ormerod (1960). The latter reported a case that had responded well after two large doses of radiation and it is possible that the case reported above may require radiation if his symptoms return.

Acknowledgements

I should like to express my thanks to Dr. C. Joiner and to Mr. R. J. Cann for permission to publish this case.

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A CASE OF ACUTE RETROPHARYNGEAL NOCARDIAL INFECTION CONTROLLED BY STREPTOMYCIN AND TETRACYCLINES

By W. STIRK ADAMS (Birmingham)

THE aerobic actinomyces were first identified as pathogenic to animals by M. E. Nocard of the Institute Pasteur in Paris in 1888, as the causal organism for farcy in oxen in Guadeloupe: two years later they were first recognized as pathogens in man by Eppinger in 1890. Although cases of this infection in human pathology have been frequently reported since its first discovery, and nocardiosis is well recognized in clinical pathology, F. Mariat of the Institute Pasteur has observed recently in an address in this country: "The infrequency with which diseases due to aerobic actinomycetes are diagnosed may be due in part to their being poorly known and therefore infrequently brought to mind in differential diagnosis."

A case of this apparently rare infection would therefore appear worthy of record, as it also illustrates a favourable response to treatment by tetracyclines, amongst a group of infections usually regarded as somewhat uncertain in their response to therapy.

The patient was a boy aged 5½ years, of mixed parentage—an Egyptian father and English mother—who was born in England and had always resided here. He was admitted as an emergency to the Children's Hospital, Birmingham, under my care on *19.10.59* after a week's illness, with an acute inflammatory retropharyngeal swelling which had failed to respond to penicillin. At the onset he developed an acute sore throat and swelling of the throat on the left side: on the following day swelling appeared on the right side also, and he was treated by his own doctor, Dr. A. A. Deiry, by daily injections of procain penicillin. In spite of this, local symptoms increased and he developed some dyspnœa especially at night, though there was no dysphagia.

A week after the onset he was referred to hospital, and on admission his temperature was normal, and he was not unduly distressed, though his pulse rate was 120 p.m.: respirations 20 p.m. There was no trismus, but a large reddened swelling was present on the posterior wall of the oropharynx pressing the soft palate forward and pus was discharging from two sinuses on its surface. There was no dyspnœa or stridor.

In a general examination a chain of enlarged glands were noted in the posterior cervical triangle on the left side. They were hard, discrete and mobile but not very tender. There was no glandular enlargement in the other triangles of the neck nor elsewhere. His ears were normal on inspection.

In his previous history he had passed through an attack of acute poliomyelitis at the age of twenty-one months which had involved his left leg, and given him a left external rectus paresis, and a left facial paresis. He had made a

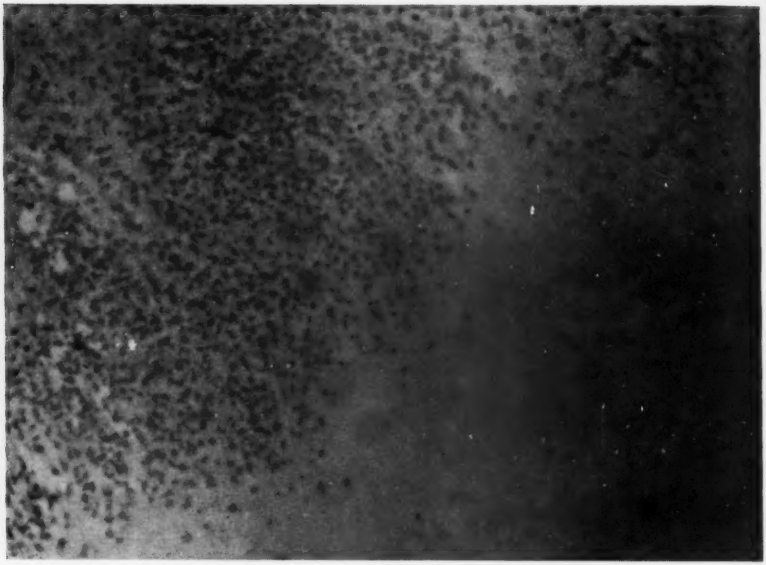
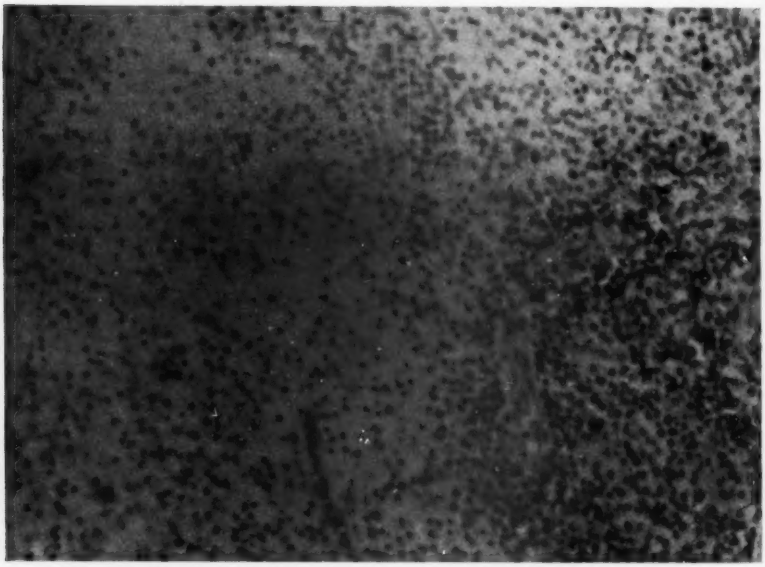


FIG. 1

(a). Showing area of necrosis bordered by cellular infiltrate. HE × 60.



(b). Cellular exudate showing histiocytes and polymorphs. HE × 125.

complete recovery and there were no sequelae. Tonsils and adenoids were removed at the age of 3½ years.

Clinically the acute condition presented as a discharging retropharyngeal abscess of unusual type—apyrexial and without dysphagia—but in view of the size of the swelling and the history of lack of response to penicillin it was decided to explore through the mouth under anæsthesia.

When the swelling was incised vertically it was found to be a mass of solid vascular tissue containing some caseous material, but there was no abscess cavity. Tissue was sent for culture and histology.

Further Investigations

The Mantoux reaction, W.R., and Kahn tests were all negative.

Dr. Astley reported the X-ray films showed no evidence of any bony lesion in the cervical spine: in the lung fields there was no evidence of active disease, though an old primary healed tubercular focus seemed probable as calcified spots were noted at the right hilum and base.

In cultures from the abscess Dr. Rogers isolated *H. influenzae* in a mixed infection but there were no hæmolytic streptococci: and Dr. Cameron reported in the histological sections:

> "A granulomatous lesion with extensive epithelial ulceration and necrosis extending deeply into the submucosa. The necrotic areas contain abundant Gram positive cocci in clumps and short chains. The surrounding tissue is heavily infiltrated with polymorphs, lymphocytes, histiocytes and plasma cells with numerous eosinophil leucocytes in some areas. There is much vascular proliferation and two small vessels show fibrinoid necrosis of the wall. There is no evidence of neoplasm or specific inflammation. Foamy macrophages as seen in scleroma are not a feature."
>
> Dr. Cameron comments:—"This is probably a severe but simple non-specific inflammatory lesion. The vascular lesions were not associated with giant cells as seen in Wegener's granuloma, but suggest the possibility of polyarteritis in the lung or elsewhere."

The diagnosis of a necrotizing ulcerative pharyngitis was made: and he suggested further bacteriological studies.

Ten days later the blood picture was: Hæmoglobin 93 per cent., white blood cells 13,000 distributed as follows:

Differential Count	*Per cent.*	*Per c.mm.*
N. polymorphonuclears (non seg)	2	260
N. polymorphononuclears (seg.)	44	5,720
Eosinophils	13	1,690
Basophils	1	130
Lymphocytes	37	4,810
Monocytes	3	390

R.B.C. normal. Platelets plentiful.

Treatment

For five days after admission he received 2 grammes of streptomycin daily, and tetracyclines 100 mg. six hourly. The latter was continued for six weeks during his stay in hospital, and Vitamin B. solution 1 drachm daily.

Progress

The local lesion produced little general disturbance and he remained well, lively and interested, feeding normally and although a mild pyrexia continued for five weeks, it only reached 100°F. on two occasions. The pulse rate however was continuously raised, ranging between 100-120 per minute. The cervical adenopathy subsided, but the large inflammatory mass persisted in the posterior wall of the oropharynx, though this did not appear to disturb him unduly. A fortnight after admission the retropharyngeal sinuses continued to discharge into his mouth, and some œdema of the surrounding tissue was still noticeable. Further cultures on *3.11.59* showed no organism of pathological significance.

A month after admission the retropharyngeal swelling was still present though smaller and less œdematous. It was then a pyramidal fibrous mass approximately 3 cm. in diameter, lying slightly to the left of the midline on the posterior wall of the oropharynx at the level of the soft palate.

In view of the slow response to treatment and the uncertain etiology, a further exploration was undertaken, and on *19.11.59* under general anæsthesia the mass was excised—in fragments—through an oral approach by a vertical elliptical incision in the posterior pharyngeal wall. There was no evidence of involvement of the deep fascia or cervical spine and the incision was closed by interrupted sutures. The specimen was divided and referred for culture and histology.

After operation he made a rapid recovery, the temperature became normal and the incision in the posterior wall of the pharynx healed. He was discharged home on *1.12.59*, six weeks after admission and has remained well since.

Reports

Specimen—retropharyngeal granuloma. Dr. Cameron reports: "A firm non-encapsulated mass of vascular tissue 1·7 × 0·8 × 0·8 cm. in maximum diameters. One surface is partly covered by a smooth pinkish white membrane with a pit in the centre. Slicing shows pale solid pinkish-yellow tissue with flecks of yellow colour."

Retropharyngeal granuloma

Two portions of tissue 0·8 and 1·0 cm. in maximum diameters. They are partly covered by a smooth pale membranous surface.

Histology

"There is deep ulceration with epithelial hyperplasia at the margins. There is acute inflammatory exudate in the ulcer crater and this is surrounded by a wide zone of granulation tissue and cellular infiltration. The chief cells are lymphocytes, plasma cells and macrophages. Adjoining arteries show marked obliterative endarteritis. No frank necrosis and no organisms are seen."

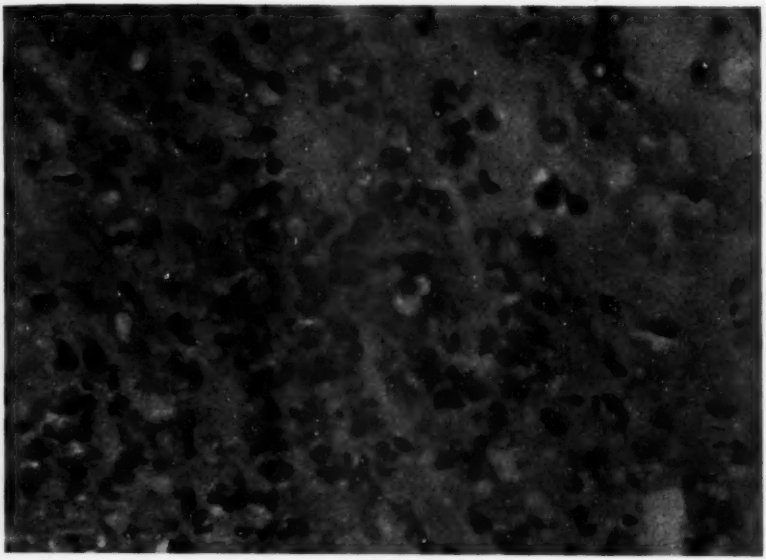
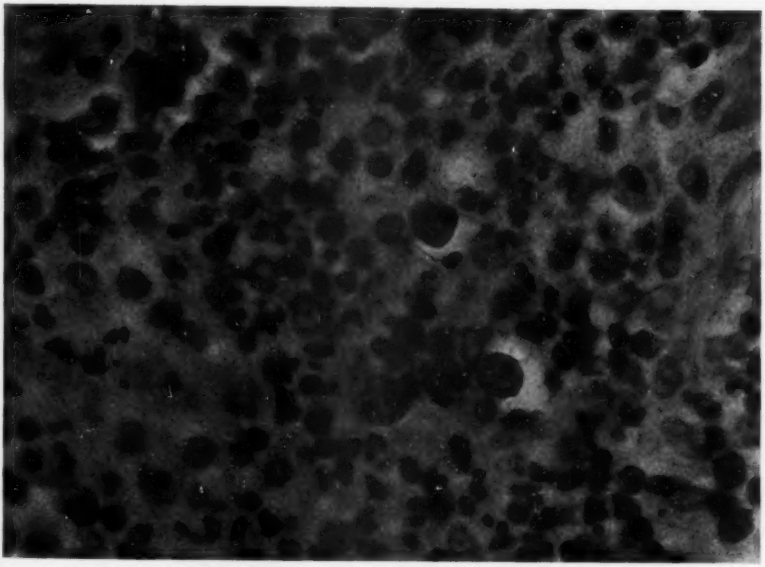


FIG. 2

(a). Perivascular and diffuse cellular infiltration. HE × 60.



(b). Detail of infiltration showing many plasma cells, some with abundant eosinophylic cytoplasm and also histiocytes and pyknotic polymorphs. HE × 125.

Diagnosis. Non-specific granuloma.

In culture Dr. Rogers recovered a *Nocardia*. Cultures on Sabouraud's medium yielded an organism which appeared to be a nocardia, and he asked for confirmation from the University of Birmingham Department of Bacteriology: Professor Shrewsbury confirmed the nocardial infection.

Further histological sections were then examined but mycelial foci were not discovered.

Annotations

In his original description of the aerobic actinomycete as the cause of the farcy infection in oxen in Guadeloupe in 1888, Nocard reports M. Couzin's description of the local pathological lesion in its more acute form as follows: "L'affection semble débuter par l'inflammation d'un ganglion lymphatique; les ganglions brachiaux, pré-scapulaires, pré-pectorauz sont les premiers et le plus fréquemment atteints; le ganglion tuméfié, chaud, douloureux, oedémateux, augmente lentement de volume et finit par offrir les caractères d'un abcès froid induré; la ponction donne issue à un pus épais, crémeux, parfois caséeux et grumeleux. La paroi de l'abcès est extrêmement épaisse (8, 10, 12 centimètres), indurée, lardacée, criant sous le tranchant de bistouri, et sa face interne est tomenteuse, douce au toucher, bourgeonneuse", this is quoted here for comparison with the clinical features of the lesion in the case presented.

The character of the pharyngeal lesion in the case presented here, had a close resemblance to that described by Couzin.

In 1890, the first report of the organism as a pathogen in human pathology was made by Eppinger who recovered it from a case of pseudo-tuberculosis with meningitis, and named it *Cladothrix asteroides*. This species was later named *Nocardia asteroides* by Blanchard in 1896.

Biologically the nocardia are members of the actinomycetes—a group of filamentous organisms which are now classified as bacteria rather than fungi, although they are usually filamentous and in this form they have a diameter of not more than 1μ.

A reclassification of these organisms was introduced in 1943 by Waksman and Henrici on cultural characteristics, and in this the term actinomycosis is reserved for the anaerobic form, of which there is only one member, *Actinomycetes Israeli*, and the aerobic forms which had hitherto been included in the genus were distributed between the streptomyces and nocardia according to whether they do or do not produce conidia in culture. (M.R.C. Memorandum No. 23 1958 H.M.S.O. Nomenclature of fungi pathogenic to man and animals).

Recorded cases of human nocardial infection in the literature indicate that these may occur either as a localized and usually subcutaneous lesion with regional adenitis, or as major systemic invasions which are most frequently pulmonary, meningeal or intracranial.

It has also been observed that as the organism in its bacillary form may be acid fast, errors in diagnosis are possible. Reporters comment that this infection may not be so rare as it has been thought to be.

The Therapeutics of the Actinomyces

In a foreword to the report of the symposium recently held under the ægis of the British Post Graduate Medical Federation at the Brompton Hospital

in *July 1957*, Dr. G. Scadding observed that the therapy of fungal infections with the exception of Actinomycoses is poorly defined, in spite of the range of agents old and new which have been brought forward for consideration. (Foreword to "Fungous Diseases and Their Treatment," Riddell and Stewart, London. Butterworth & Co., 1958.)

The treatment of *Actinomyces Israeli*—the anaerobic group—by massive doses of penicillin up to 6 million units daily, is described on p. 226 of this report by O. S. Tubbs and the treatment of infections due to aerobic actinomycetes by F. Mariat of the Institute Pasteur, Paris, on p. 121, who concludes that sulphonamides are frequently inhibiting to pathogenic species of nocardia and streptomyces.

In the case presented treatment was empirical as the infecting organism was not identified until after his recovery. The initial streptomycin therapy was given as a protection against a Sonne infection in the hospital ward, and tetracyclines as a broad spectrum antibiotic until the causal organism could be identified.

Although the response of the lesion to the antibiotics was slow, after a month's treatment the local lesion was considerably reduced in size with diminution of surrounding œdema, and the enlargement of the cervical glands disappeared. It would also seem probable that the rapid healing of the pharyngeal incision after excision of the mass was a result of the antibiotic control of the infection.

It has not been possible to report the species of nocardia in this infection as detailed investigations were not undertaken as the patient had recovered: and the portal of entry remains obscure, though from the history of the illness it would seem probable it had its origin in the nasal mucosa or in the post nasal space with secondary invasion of a left retropharyngeal lymphatic gland.

Acknowledgements

My thanks are due to Dr. A. H. Cameron and Dr. K. B. Rogers in the Department of Pathology and Bacteriology in the Children's Hospital, Birmingham, for their help in the identification of this unusual infection and also to Professor J. F. D. Shrewsbury, for his opinion: also to Dr. R. Astley for the radiographic studies and to the University of Liverpool Medical Library for access to the original article by M. E. Nocard in the Annales de L'Institut Pasteur, for the University of Birmingham Medical School Library.

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81 Harborne Road,
Edgbaston, Birmingham

SOCIETIES' PROCEEDINGS

THE SCOTTISH OTOLARYNGOLOGICAL SOCIETY

THE eighty-first (Jubilee) meeting of the Society was held in Edinburgh Royal Infirmary on Saturday, November 12th, 1960, the President, Dr. Forbes M. Walker in the Chair.

Dr. Douglas Guthrie read a paper on the history of the Society entitled "Looking back on fifty years", in which he traced its progress since the initial meeting in Edinburgh Royal Infirmary on November 11th, 1910. He mentioned many of the personalities prominent in the specialty and in the affairs of the Society in these early days and showed a series of lantern slides depicting the early Chairmen and some of the presidents. He pointed out that of the thirty-one original members who formed the Society in 1910 only one was still with us, and he expressed pleasure that Dr. R. P. Mathers of Dundee had consented to be the next President during the Jubilee Year.

Following Dr. Guthrie's paper a short clinical part of the meeting involved the demonstration and discussion of twelve cases and some exhibits.

Dr. A. Brownlie Smith showed a case of otosclerosis where different methods of stapedectomy and reconstruction were used in each ear owing to the different pathology around the two footplates, and in the subsequent discussion Dr. A. Gibb stressed the need for a detailed description of the microscopic appearances in this region in the records of all cases in order that the best procedure in different types of the disease may be determined. Dr. Smith also showed a case of carcinoma occurring in a radical mastoid cavity, treated with the linear accelerator, and it was pointed out in discussion that these cases did not seem to be as rare as might be thought and that the possibility should be borne in mind when granulations developed.

A further patient demonstrated by Dr. Smith suffered from recurring carcinoma of the larynx for which laryngofissure had been carried out twice since 1946. Keratosis of the reformed cord had developed into carcinoma, and after a further recurrence he was now being treated by radiotherapy. In the ensuing discussion Dr. I. M. Farquharson pointed out that in many cases where malignancy develops from keratosis irradiation seems to be detrimental, and he recalled such a case where radiotherapy was followed by a very rapid increase in the growth and the glands.

Dr. Farquharson showed a case of leucoderma affecting the vestibule of the nose and the discussion centred round the possible cause of the condition. Dr. Brownlie Smith suggested, in view of a previous case which he had known, that the use of a paraffin ointment might be responsible and he had elicited the fact that the present patient had been in the habit of using a considerable amount of Mentholatum.

A case of otosclerosis treated by stapedectomy followed by a fat graft and

steel wire prosthesis was shown by Dr. B. H. Colman. He pointed out that the procedure had certain advantages over the more usual vein graft operation. It was technically easier and experimentally the fat was associated with less scarring round the oval window than the vein grafts. The steel wire was also easier to use than polythene in cases where the oval window was partially obscured.

Dr. Colman also showed a case of atrophic rhinitis where nasal narrowing had been carried out by inserting acrylic plaques by the sublabial approach. Various members contributed to a discussion on the different methods of treatment which might be tried in this condition, and Mr. R. Scott Stevenson reported an outstanding improvement in three such cases following the topical instillation into the nose of hydrocortisone and chloromycetin eye drops.

Dr. R. B. Lumsden showed the prototype of a new apparatus for ultrasonic therapy in Ménière's disease, and also a series of coloured slides to show the changes in the labyrinth of cats at different stages of treatment.

Dr. Colman showed a series of photomicrographs to illustrate the behaviour of various types of graft tissue in the oval window, the results of which had led him to think that fat was the tissue of choice.

Following the clinical part of the meeting Dr. R. P. Mathers of Dundee, an original founding member, was elected President of the Society. In the evening a Commemorative Dinner in the Royal College of Surgeons of Edinburgh was attended by members and their guests. Professor John Bruce, C.B.E., P.R.C.S.Ed., proposed the health of the Society and Dr. Mathers replied. Among the guests were Mr. J. P. Monkhouse and Mr. J. H. Otty, Presidents of the Sections of Otology and of Laryngology of the Royal Society of Medicine, and Mr. T. G. Wilson, P.R.C.S.I.

GENERAL NOTES

VIIème CONGRÈS INTERNATIONAL O.R.L.

Le VIIème Congrès International O.R.L. se tiendra du 23 au 29 juillet 1961 à Paris, à la Nouvelle Faculté de Médecine, Rue des Saints-Pères, sous la présidence de M. le Professeur Aubry.

Trois très importants rapports seront discutés:

1°) Les acquisitions récentes en pathologie et en thérapeutique de l'otosclérose.
Rapporteurs: MM. les Professeurs Sourdille, Sercer and Krmpotic, Weber, Ardouin, Larroude, Ferreri and Schambaugh.

2°) Indications et résultats après 5 ans, de la chirurgie et des associations radio-chirurgicales dans le traitement des cancers du larynx et de l'hypopharynx.
Rapporteurs: MM. les Professeurs Pietrantoni and Agazzi, Alonso, Casadesus, Chevalier-Jackson, Leicher, Leroux-Robert, Ormerod.

3°) Rapports entre l'allergie et les infections du nez et des bronches.
Rapporteurs: MM. les Professeurs Van Dishoeck, Clerici, Hlavacek, Voohorst, Mayer, Laskiewicz, Teracol.

De nombreuses réunions et festivités sont envisagées pour les Congressistes et leurs épouses. A la suite du Congrès, des excursions auront lieu; elles seront proposées dans un avant-programme et réalisées par les soins des Wagon Lits Cook, 22 rue de l'Arcade, Paris, qui sont notre agent officiel habilité pour les transports et réservations d' hôtels.

Pour tous renseignements, écrire au Dr. Henry Guillon, Secrétaire Général, 6 avenue Mac-Mahon, Paris.

THE INSTITUTE OF LARYNGOLOGY AND OTOLOGY

330-332, Gray's Inn Road, London, W.C.1.

SPECIAL LECTURES

At an advanced level, open to all members of the Specialty and to post-graduate students (whether enrolled students of the Institute or not).

Saturday, 6th May, 1961. Symposium beginning at 10 a.m.

Professor Otto Löwenstein, Birmingham. "The evolution of static and acoustic sense organs."

Dr. Hallowell Davis, St. Louis. "The relation of presbyacusis to noise-induced hearing loss."

Dr. Jürgen Tonndorf, Iowa City. "Cochlear hydrodynamics."

General Notes

GUEST LECTURE

Thursday, 18th May, 1961, at 5.30 p.m.

Dr. J. L. Stafford, M.B., Ch.B., M.R.C.S., L.R.C.P., St. George's Hospital, London. "Modern trends in the theory and practice of blood transfusion."

NON-MEDICAL COURSES

31st May, 1st and 2nd June, 1961—Hearing and mental assessment of the deaf child—for psychologists.

The number of entries is limited in each course. A detailed syllabus of the above can be obtained on application from the Secretary-Administrator.

TEMPLE UNIVERSITY SCHOOL OF MEDICINE

PHILADELPHIA 40. PA.

A "Short course in Audiology" is offered several times each year by the Section of Audiology, Department of Otorhinology, Temple University School of Medicine, Philadelphia 40, Pa.

The course will be one week in length and will encompass theoretical and clinical aspects of audition. Hearing testing and interpretation will be stressed. Office procedures, equipment and Industrial Audiometry will be discussed.

Further information may be obtained by writing to Philip E. Rosenberg, Ph.D., Director, Section of Audiology, Temple University Medical Center, 3401 North Broad St., Philadelphia, Pa.

ROYAL COLLEGE OF SURGEONS
OTOLARYNGOLOGY LECTURES

THE following lecture, arranged jointly by the Royal College of Surgeons of England and the Institute of Laryngology and Otology, will be delivered in the Lecture Hall of the College on Thursday afternoon at 5.30 p.m.

1961

May 4th. DR. R. BODLEY SCOTT. "Blood diseases in relation to Otolaryngology."

THE INTERNATIONAL COURSE IN PAEDO-AUDIOLOGY

JUNE 13TH-16TH, 1961

It is intended to organize at Groningen University, The Netherlands, a second international course in paedo-audiology, in close co-operation with the

Royal Institute for the Deaf and the Institute for the Hard of Hearing, Groningen. As was also the case in the first course, which was held in 1953, the problem of the hearing handicapped child will be the feature of the programme. Special emphasis will be laid on the educational treatment of young children.

The course, which will take place from June 13th to 16th, is subsidized by the World Health Organization and Unesco. The official language is English. The subscription is 60 Dutch guilders. Those who are interested in participating in the course are requested to send their application to the Audiology Department of the University Hospital, Groningen.

Participants are requested to book hotel accommodation at their earliest convenience. The course fee may be sent by bank transfer to "Mesdag en Groeneveld's Bank, 17 Oude Boteringestraat Groningen" in favour of "International Course in Paedo-Audiology".

INTERNATIONAL SYMPOSIUM ON AUDIOSURGERY

The National School of Medicine and Work, the National Institute of Medicine, and the Centre of Surgical Specialities have organized an International Symposium on Audiosurgery in Madrid in August 1961, on 2nd, 3rd and 4th. The Chairman will be, Dr. Francisco Antoli-Candela.

The subjects to be considered and discussed will be the Audiosurgery of the congenital defects of the ear, the chronic suppurative processes of the ear and its sequelae, and otosclerosis. The surgery of the facial nerve will also be considered. There will be surgical sessions and simultaneous translations.

An official lunch has been arranged and will be included in the subscription to the Symposium. On the 5th August there will be a visit to the "Escorial" and to the "Valle de las Caides", and a "fiesta Campera". A sight seeing programme for the participants and their guests who wish to see the surroundings of Madrid, the South of Spain, and Palma de Mallorca has also been organized.

For further information please write to:

Secretaria General, Dr. A. Juderias, Centro Nacional de Espedialidades Quirurgicas, Ciudad Universitaria, Madrid, Spain.

THE COLLEGE OF SPEECH THERAPISTS

The College of Speech Therapists is holding a Conference in Birmingham from 24th to 28th July, 1961.

Abnormalities of speech and language will be presented as systemic disorders under the guidance of the concepts of Signs, Signals and Symbols.

For further information please write to the Conference Organizing Secretary, 16 York Road, Birmingham 16.

General Notes

BACK JOURNALS REQUIRED AND FOR SALE

If any subscriber can spare copies of the January 1945 and June 1950 issues of the Journal of Laryngology & Otology they would be very much appreciated by the Librarian of The National Institute for the Deaf, 105 Gower Street, London, W.C.1.

If anyone would like to buy a set of the journals from 1921 to 1960 (mostly bound volumes), and the two relevant indexes etc., please apply to Donald G. P. Macalister, 5 Clarendon Place, Leamington Spa.

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